JOURNAL
of the
American Veterinary Medical Association

E. A. RODIER, Acting Secretary-Editor

H. D. BERGMAN, President, Ames, Iowa. M. JACOB, Treasurer, Knoxville, Tenn.

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WHAT OF THE FUTURE?

An editorial has been defined as an expression of the editor's viewpoints and policies, as well as those of the organization his publication represents.

This editorial has a two-fold purpose. First, to endeavor to promote a wider and more intense interest in the problems confronting your Association and, secondly, to give the membership a basis for judging the man, recently selected as Acting Secretary-Editor by the Special Committee of the Executive Board, who is practically unknown to most of you because of having spent the past ten years in the Orient.

First of all, you perhaps will want to know something about the qualities which go to make up the personality of the man who is being considered for the executive office of your Association. The definite qualities one must possess to be a successful executive have been reduced to a science. Many articles enumerating these qualities have been written. One by Mathew S. Sloan, president of the Brooklyn-Edison Company, which appeared in *The Magazine of Business*, lists these qualities as follows: Personality, reputation for square dealing, courtesy, tact, tolerance, skill, promptitude, a coöperative spirit and a desire to please. It would seem that these qualities are as desirable in an executive in our national association as in any other type of organization.

All of these attributes are necessary to render that intangible thing called service, which should include readiness to go beyond the mere letter of an agreement, expressed or implied. Your Acting Secretary-Editor pledges to observe these rules of conduct, realizing that fostering good will among all is a permanent asset to the Association.

It would seem that service is an essential prerequisite to any effective plan of

reorganization. This short editorial does not permit a complete description of all of the author's ideas regarding the subject of reorganization, but perhaps it would not be taken amiss, if the thought is presented that every successful association is so organized as to include in its membership roll every member of local organizations. As far as the writer knows, this idea has not been previously emphasized as an essential factor in uniting and thereby building up the veterinary profession to the point where it will be given the recognition which it now demands, but so far has been unable to secure to the fullest extent.

The type of service rendered by a national association is determined by its strength to obtain worthwhile advantages. This strength can only be obtained through unified action. How far we travel on the road toward this goal will depend upon how carefully the members of state organizations select their delegates and alternates to the national convention each year. Upon them depends the type of representation which determines the future welfare of the A. V. M. A. and the profession.

In unity there is strength. We must find a way to acquire this unity, even though it is a recognized fact that there are many widely divergent groups with conflicting ideas and interests which make up the American Veterinary Medical Association. It would seem that the veterinary profession is now approaching a fork in the road. If we continue to follow the path of disunity, the profession is bound to gradually disintegrate, as well as your national organization. Whereas, if we follow the path of national unity, we are bound to achieve that high place among the medical professions to which we aspire.

The writer takes full responsibility for the ideas expressed herein and sincerely hopes that they meet with the general approval of the JOURNAL readership. Your reaction to these ideas would be appreciated, for it is only by knowing majority sentiment that policy can be moulded to please the largest number of members.

E. A. R.

PROFESSIONAL GOODFELLOWSHIP

For some 420 young men, the cream of our 1938 veterinary graduates, a new way of life has begun. Four years and more of happy, care-free college life, with its trials and its triumphs, its hopes and its ambitions, have passed into history. Only the hopes and ambitions remain.

How vivid are the memories that we "old timers" recall of those first few months and years in the practice of our beloved profession! We were the fortunate few who, often apparently by chance, came under the influence of professional men who believed in and practiced the Golden Rule. We were indeed fortunate, for through the aid and kindly guidance extended to us, we were enabled to attain our ambitions.

But many of us were forced to face a stern world of cold realities without benefit of the experience necessary to form sound judgments in the art of human relationships so essential to attain success. Therefore, some of us wasted many precious years in learning the way to promotion and pay and many others of us fell by the wayside, lost talent that might have been saved for the profession by the right word spoken at the right time.

Professional goodfellowship pays big dividends, not only to the one to whom it is extended, but also to the profession as a whole, and in personal satisfaction to the man who extends it. The first step in the right direction is to persuade the recent graduate to join his local and national veterinary medical association.

"Bread cast upon the waters will return a hundredfold." E. A. R.

Inscription on a stone in the Hartsdale, N. Y., dog cemetery: "Jack, as ever, precedes his master by a few steps."—Joseph Mitchell, My Ears Are Bent (Sheridan House), *Readers' Digest*.

Case Reports

One of the principal objections made to our former style of editing the JOURNAL was the paucity of clinical and case reports that it contained. Your acting editor recognizes the justice of your criticisms and, for his part, is more than anxious to please the practitioners who want this type of material.

By way of explanation, it is not so much the cases that present rare and unusual features, interesting as they are, that are of real value to the clinician, but the ordinary run of everyday cases that come before your attention. You know, the type that you gossip about before and after a local meeting. This is the sort of thing that would be particularly interesting and valuable to the members at large, especially if accompanied by pictures.

Your published reports will benefit other clinicians but, you in turn, will be repaid for your efforts by learning about the experiences of others. May we expect your coöperation in this important matter?

Congratulations

The veterinary profession of the United States, as represented by the American Veterinary Medical Association, desires to extend hearty congratulations and sincere appreciation to their British colleagues for the exceptionally fine manner in which they have rendered tribute to Sir John M'Fadyean, whom we believe is admired fully as much on this side of the Atlantic Ocean as he is in his home country. We heartily subscribe to the sentiment expressed in the preface of the December issue of the Journal of Comparative Pathology and Therapeutics that, "When a man has rendered what is clearly perceived to be a signal service through his own efforts and initiative, to the lasting good of his fellow man, it would seem that nothing should deter giving expression, on fit and proper occasion, within his lifetime to the natural, human response of gratitude." Acting upon that premise, Sir John's colleagues prevailed upon him to turn over the preparation of the Jubilee number to them. To them, we say, "Well done!" To Dr. John R. Mohler, our own beloved Chief of the United States Bureau of Animal Industry, we extend thanks for his contribution to this number, rendered in his usual masterly literary style.

Equine Encephalomyelitis

Veterinarians and livestock men alike are becoming gravely concerned over the increasing number of "sleepy horses," in Illinois, Michigan and Iowa, particularly, which the practitioners of those states are being called upon to handle.

In the state of Illinois alone, 16 counties are now included in the stricken areas. On August 15, there were more than 75 horses reported effected with the malady.

Since the first equine encephalomyelitis outbreak in California in 1932, this disease has been spreading eastward at an alarming rate. Large-animal practitioners have a real problem on their hands in finding ways and means to effectively prevent the further spread of this disease.

Reciprocity

In *The Iowa Veterinarian*, May-June, 1938 number, appears an editorial on the subject of patronizing biological and pharmaceutical firms that advertise in our publications. That is good, sound, common sense. Our advertisers, as Dr. C. J. Scott points out, are rendering assistance to clinicians in many ways besides restricting sales to ethical veterinarians. To name but one example, how many practitioners have received courtesy diagnosis aid from commercial houses? Let's show them that we appreciate their efforts by restricting purchases to our advertisers.

Secretaries, Attention!

The JOURNAL needs more reports of association meetings, personals and other items of local interest than are now being received. May we ask your cooperation in promptly supplying us with this material, together with advance notices of coming meetings?

Coming Veterinary Meetings

Ak-Sar-Ben Veterinary Medical Association. Fontenelle Hotel, Omaha, Neb. September 12, 1938. Dr. J. D. Ray, Secretary, 1124 Harney St., Omaha, Neb.

Willamette Valley Veterinary Medical Association. Gresham, Ore. September 14, 1938. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Kansas City Veterinary Medical Association. Kansas City, Mo. September 19, 1938. Dr. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego Veterinary Medical Association. Zoölogical Research Building, Balboa Park, San Diego, Calif. September 19, 1938. Dr. E. F. Sheffield, Secretary, 3895 Pacific Blvd., San Diego, Calif.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. September 21, 1938. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Southwestern Minnesota Veterinary Medical Association. Lakefield, Minn. September 27, 1938. Dr. Louis E. Stanton, Secretary, Jackson, Minn.

New England Veterinary Medical Association. Concord, N. H. October 3-4, 1938. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. October 4, 1938. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. October 6, 1938. Dr. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. October 5, 1938. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Illinois Veterinary Conference, University of. University of Illinois, Urbana, Ill. October 5-7, 1938. Dr. Robert Graham, University of Illinois, Urbana, Ill.

Houston Veterinary Association. Houston, Texas. October 6, 1938. Dr. E. G. Pigman, Secretary, 4206 Polk Ave., Houston, Texas.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. October 11, 1938. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

West Virginia Veterinary Medical Association. Kanawha Hotel, Charleston, W. Va. October 11-12, 1938. Dr. J. H. Rietz, Secretary, Oglebay Hall, West Virginia University, Morgantown, W. Va.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. October 12, 1938. Dr. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. October 12, 1938. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Eastern Iowa Veterinary Association, Inc. Hotel Montrose, Cedar Rapids, Iowa. October 18-19, 1938. Dr. H. E. Tyner, Secretary, New London, Iowa.

Purdue University Veterinary Short Course. Purdue University, Lafayette, Ind. October 18-21, 1938. Dr. R. A. Craig, Department of Veterinary Science, Purdue University, Lafayette, Ind.

International Association of Milk Sanitarians. The Allerton, Cleveland, Ohio. October 19-21, 1938. Mr. C. Sidney Leete, Secretary, Department of Health, Albany, N. Y.

Interstate Veterinary Medical Association. Warrior Hotel, Sioux City, Iowa. October 20-21, 1938. Dr. W. A. Aitken, Secretary, Merrill, Iowa.

Florida State Veterinary Medical Association. Miami, Fla. October 24-25, 1938. Dr. J. V. Knapp, Secretary, The Capitol, Tallahassee, Fla.

APPLICATIONS FOR MEMBERSHIP

The month of September marks the opening of the fall term for most veterinary colleges. This issue of the JOURNAL is therefore dedicated to all veterinary students, and particularly to members of the Junior A. V. M. A., selected for high standing. To them, hearty congratulations and a warm welcome is extended with the hope that many more students will qualify for membership during the school year just beginning. Seniors, we are looking forward to the pleasure of seeing your names on our list of members next June.

FIRST LISTING

CHRISTOFFERSON, FRANKLIN F.
Snohomish, Wash.
B. S., D. V. M., State College of Washington, 1937. Vouchers: E. E. Wegner and J. E. McCoy.

COPE, RUSSELL P.
1205 San Pablo Ave., Berkeley, Calif.
D. V. M., Kansas State College, 1936. Vouchers: S. T. Michael and John McInnes.

GOCHENOUR, RAYMOND B.
705 Varnum St. N. W., Washington, D. C.
V. M. D., University of Pennsylvania, 1937. Vouchers: W. S. Gochenour and H. W. Schoening.

GOCHENOUR, WILLIAM S., JR.
705 Varnum St. N. W., Washington, D. C.
V. M. D., University of Pennsylvania, 1937. Vouchers: W. S. Gochenour and H. W. Schoening.

JUNG, OTTO E.
1417 E. 54th St., East Saint Louis, Ill.
D. V. S., Kansas City Veterinary College, 1908. Vouchers: Charles Barnes and G. H. Bruns.

OSTERHOLTZ, WILBERT E.
4332 Hartford St., Saint Louis, Mo.
D. V. M., Kansas State College, 1935. Vouchers: F. R. Koutz and S. T. Michael.

RACOFF, HERBERT
408 State Office Bldg., Columbia, S. C.
D. V. M., Cornell University, 1937; M. S., Ohio State University, 1938. Vouchers: W. A. Hagan and Leonard W. Goss.

SPLAVER, DAVID
241 E. Highland Ave., Tracy, Calif.
D. V. M., Ohio State University, 1937. Vouchers: J. F. Claire and S. T. Michael.

Applications Pending

SECOND LISTING

(See August, 1938, JOURNAL)

Courter, Robert D., 10 S. Belvidere St., Richmond, Va.
Curtis, Robert, 422 Edgewater Pl., Portage, Wis.
Davis, John A., Buxton, N. Dak.
Deming, David F., 123 Andrews St., Massena, N. Y.
Deubler, Josephine, Newton, Pa.
Dickman, Andrew J., Box 622, Elko, Nev.
Elliott, Edward W., Park River, N. Dak.
Essex, John J., U. S. Quarantine Sta., Rosebank, L. I., N. Y.
Field, Lincoln E., Middleburgh, N. Y.
Fitzgerald, Francis J., 376 Lunenburg St., Fitchburg, Mass.
Fleenor, Walter, Fairmount, N. Dak.
Fortune, Steven G., 39 Livestock Exchange Bldg., Wichita, Kan.
Franks, Robert D., 769 Vance Ave., Memphis, Tenn.
Frazee, James A., R. 1, Annandale, N. J.
Greenlee, Lt. Col. Christian W., Governors Island, N. Y.
Grodin, Irving R., Box 844, San Juan, P. R.
Haley, John S., 1128 Kansas Ave., Topeka, Kan.
Haubrich, Wilson R., Claremont, N. H.
Hester, Harold R., University of Illinois, Urbana, Ill.
Hillstrom, Werner F., 825 Bond Ave., Collinsville, Ill.
Hofstrand, Carl H., Churchs Ferry, N. Dak.
Holt, Alfred L., 233 Rodes Ave., Lexington, Ky.
Horn, Wiley H., 1627 Grand Ave., Fort Worth, Texas.
Hughes, Leland S., 1327 8th Ave., Worthington, Minn.
Jackson, Guy S., 118 W. Church St., Frederick, Md.
Jacobs, Joseph, 4910 15th Ave., Brooklyn, N. Y.
Kritt, Abraham A., 429 First Ave., Albany, Ga.
Leeson, John E., 11-23 Saint Albans, Toronto, Ont.
Long, Ray S., Upham, N. Dak.
Lutvack, Harry A., 2194 Barnes Ave., New York, N. Y.
Massinger, Wesley, Chalfont, Pa.
Montgomery, Lancot R., Casselton, N. Dak.
Montgomery, Roscoe G., Cogswell, N. Dak.
Moore, Edward C., 52 Pleasant St., Waterville, Maine.
Nicholson, Lyle G., Western Washington Exp. Sta., Puyallup, Wash.
Osteen, Oswald L., Box 83, Beltsville, Md.
Owens, F. Herbert, Jr., 36 White House Pike, Audubon, N. J.
Prchal, Charles J., 5434 S. 22nd St., Omaha, Neb.
Rudman, Henry, 1915 Morris Ave., Union, N. J.
Schnaas, William, Motolinia No. 2, Mexico, D. F., Mexico.
Schutz, Frederick W., Brewster, N. Y.
Smith, Connor D., Standish, Mich.
Sutton, James M., Sylvester, Ga.
Thomas, George O., Jr., 769 Vance Ave., Memphis, Tenn.
Van de Erve, Jacob, 222 8th Ave. S. E., Minot, N. Dak.
Volkmar, Fritz, 1935 Irving Park Blvd., Chicago, Ill.
Walker, Earl C., 1110 7th Ave. S., Fargo, N. Dak.
Wermuth, John J., R. 1, Box 200, Western Ave., Albany, N. Y.
Williams, David M., McComb, Miss.
Williams, Theron, Box 844, San Juan, P. R.
Woodcock, Jorgan G., Walken Farm, Mount Kisco, N. Y.

The amount which should accompany an application filed this month is $6.67, which covers membership fee and dues to January 1, 1939, including subscription to the JOURNAL.

Cultivation of Pigeon-Pox Virus on the Chorio-Allantoic Membrane*

By F. R. BEAUDETTE *and* C. B. HUDSON
New Jersey Agricultural Experiment Station
New Brunswick, N. J.

Several viruses of avian origin have already been cultivated on the chorio-allantoic membrane of the developing egg. In order to propagate a virus in this manner, the inoculum must, or at least should, be free of bacterial contamination. In such septicemic diseases as fowl plague, a bacteria-free virus is easily obtained from the blood. In other diseases, the virus is localized in some part which is naturally contaminated with bacteria. Laryngotracheitis and bronchitis are examples of this type of infection, but even here a pure virus is easily obtained by the simple process of filtration. However, in the pox diseases, the viruses are not only contaminated with bacteria, but they are exceedingly difficult to filter.

Woodruff and Goodpasture[1] have described three methods for obtaining non-contaminated pox virus. In one of these, the feathers are plucked from the head of a one- to two-week-old chick and virus is inoculated at three points 1 cm apart. On the sixth or seventh day after inoculation, at which time nodules are likely not to be invaded by bacteria, the chick is killed. The head is bathed with 95 per cent alcohol and the nodule is cut off with a sterile knife, deep enough to include the infected cores. Then, with sterile instruments, the infected core is forced out of the follicle from the cut surface. If a piece of the infected core proves sterile when planted in glucose-yeast broth, the other piece is used as a source of virus.

In the second method, seven- to ten-day-old lesions are subjected to digestion in a 1 per cent trypsin solution to free the virus bodies. Then, after several washings in sterile saline, a single virus body is picked up in a minute sterile pipette of the Chambers microdissection apparatus. In the third method, pox lesions are freed of tissue by tryptic digestion and the resulting virus bodies are washed several times and then treated for one day with 1 per cent potassium hydroxide solution to kill contaminating organisms.

As pointed out by these authors, treatment with potassium hydroxide solution without previous digestion in trypsin fails to render the material free of certain moulds and occasionally a bacillus. Such was our experience, and since we had no trypsin at the time, a modification of the first method was selected as the one most likely to succeed. The results were satisfactory in the case of fowl-pox, but were of no value in the case of the pigeon virus.

Isolation of Virus

When the feather follicles on the leg of a half-grown chicken are inoculated with fowl-pox virus, pure virus can be scraped from the flesh side of the skin and inoculated directly into eggs. Thus, the inoculated bird has to be killed when lesions develop and the leg removed, plucked free of feathers, and singed carefully. In an atmosphere free of dust, the skin is carefully raised from the leg and held by an assistant while the deeper portions of the lesions are scraped with a sterile blood-lancet or similar instrument. The small amount of material obtained in this manner is inoculated directly into the chorioallantois with a sterile platinum needle. Some of the material may be smeared on an agar plate as a control, but contamination can be detected by cultures from the inoculated egg at the time of harvest. However, when this method was attempted with pigeon virus, it always gave heavy contamination. It appears that pigeon virus has a special affinity for follicular cells and that these always become invaded by bacteria. At-

*Journal Series paper of the New Jersey Agricultural Experiment Station, department of poultry husbandry. Received for publication, July 26, 1937.

tempts to use very young lesions gave no better results. Burnet[2] states that he has grown one strain of pigeon-pox virus on the chorioallantois but he does not describe how a pure virus was obtained to initiate the growth.

After repeated failures to obtain a pure virus from deep scrapings, it was decided to attempt filtration. Infected skin from the leg of a chicken inoculated with pigeon virus was ground with sand, centrifugalized, and passed through each of two Berkefeld-V filters. Before the skin was removed, a final attempt was made to get a pure virus by light scraping, but of the four eggs inoculated, none showed growth, nor were they contaminated. Filtrate A was inoculated into three embryonated eggs, 15, 13, and nine days old, respectively, on March 30, 1936. The sterility test on the filtrate showed contamination and the embryos were found to be dead after incubation for four, five, and nine days, respectively. Smears from the membranes showed contamination. However, the last two eggs showed definite evidence of virus growth. Filtrate B was inoculated into three nine-day-old embryonated eggs. Here again the filtrate proved to be contaminated when planted in broth. But, of the three eggs inoculated, one died after incubation for five days and showed no infection. The second, dead after incubation for nine days, showed generalized infection, but a culture from the membrane gave two colonies on the plate. The third egg was opened after eight days of incubation while the embryo was alive. The membrane showed generalized infection and the sterility test showed no bacterial contamination. Hence it appears that in this case the few bacteria that passed the filter were destroyed by the bactericidal substances in the egg. The membranes of this egg were used to initiate subsequent passages, the data on which are given in table 1. The table indicates the generation, the dates initiated, and age of the eggs at the time of inoculation. There is also indicated the number of days the eggs that supplied inoculum were incubated. If membranes of these eggs were held in the freezer, the length of time is indicated. For example, the membrane that was used to initiate the second generation came from an egg incubated eight days after inoculation, and the membrane was frozen one day before it was used as inoculum. The few embryos that died in the first 24 hours after inoculation are not considered.

SERIAL PASSAGES

The harvesting of membranes and the preparation of inoculum were always carried out in an atmosphere free of dust. In preparing the inoculum, a membrane was ground in a sterile mortar and suspended in 5 cc of broth. Only membranes of live embryos were used as inoculum. After centrifugalization, the supernatant fluid was drawn into a syringe. The chorioallantois was usually inoculated at three points with a total dose of 0.6 cc. After a series of eggs had been inoculated, about 0.6 cc of the inoculum was smeared over an agar plate as a sterility test. Likewise, at the time of harvest, a culture was made of each membrane.

Unless otherwise stated, the embryos were alive at the time of harvest. Exclusive of early deaths, table I shows that death of the embryo occurred in only five cases. Since a relatively large number of eggs was dealt with, this may be accepted as a normal mortality and not attributable to a lethal effect of the virus. The results show that the virus remains active when held as long as 38 days in a frozen state.

The lesions provoked by the pigeon virus are no different in appearance from those of fowl virus. Sometimes the lesion consists of nodules at the points of inoculation, or, if the inoculum is richer in virus, the lesions coalesce to form a thick disc at that pole of the egg. The most extensive involvement is seen when the inoculum is rich and the egg is incubated more than five days.

COLLATERAL PASSAGES

In addition to the 19 consecutive passages listed in table I, several collateral inoculations were made. A membrane from an egg of the sixth generation was used to inoculate five thirteen-day-old eggs on June 8. These were not opened until June 15,

that is, on the 20th day of incubation. Some of the eggs were already pipped. Thickening of the membrane in two cases was the only change noticed. The inoculum was only 13 days old and must have been active, because the inoculum for the seventh serial generation had been held 38 days and was active. One of these membranes was immediately used as inoculum for seven other eleven-day-old eggs on June 15, but no infection was seen in the five which survived incubation for seven days. Another membrane held twelve days and, when inoculated into 17 eleven-day-old eggs, also failed to produce visible lesions in any of them. However, one membrane was slightly thickened and, after being held in a frozen state for 46 days, was tested by the inoculation of three nine-day-old eggs on August 18. One embryo died immediately. The two others were incubated for six and eight days, respectively, without showing infection. Thus, it would appear that membranes removed from eggs near the hatching date contain little or no virus.

The potency of membranes decreases somewhat by holding in a frozen state. A heavily infected membrane of the 13th generation was held 39 days and inoculated into 23 twelve-day-old eggs in about one-half the usual dose. Of the 18 eggs that were incubated five days after inoculation, none showed infection except at the points of inoculation.

NEUTRALIZATION TESTS

Three neutralization tests were attempted. An amount of serum from an im-

TABLE I—*Data on serial passage of pigeon-poxvirus.*

GENERATION	DATE OF INOCULATION	AGE OF EGGS WHEN INOCULATED (DAYS)	INOCULUM		EGGS INOCULATED	EARLY DEAD	REMAINING	DAYS INCUBATED	RESULTS
			DAYS EGGS INCUBATED	DAYS HELD IN FREEZER					
2	4– 8–36	9	8	1	2	0	2	8	Generalized infection in both
3	4–16–36	11	8	0	4	1	3	6	One localized, two generalized
4	4–24–36	10	6	2	3	1	2	6	Localized infection in both
5	5 –8–36	10	6	8	6	0	6	7	One embryo dead on 5th day—no infection. One localized infection and four, generalized infection
		11	6	8	4	0	4	7	Good generalized infection in all
6	5–18–36	8	7	3	3	0	3	8	Generalized infection in one, localized infection in two
		11	7	3	3	0	3	8	Fair generalization in one, localized infection in one, and one showed no infection
7	7 –3–36	11	8	38	8	0	8	7	One embryo dead, all showed generalized infection
8	7–10–36	10	7	0	10	0	10	7	One embryo dead on 5th day, one dead on 7th. All showed good generalized infection
9	8 –1–36	10	7	15	6	0	6	6	Secondary lesions only near points of inoculation
10	8–22–36	10	6	15	3	1	2	6	Secondary lesions only near points of inoculation
11	9 –2–36	12	6	5	7	2	5	6	Generalized infection in one. Secondary lesions near points of inoculation in others
12	10 –1–36	12	6	23	4	1	3	5	Infected disc at inoculation pale in one. Secondary lesions near points of inoculation in two
		11	6	23	1	0	1	5	Large infected disc at inoculation pale
13	10–26–36	11	5	20	7	3	4	5	Whole membrane infected in one, good generalization in others
14	11–16–36	9	5	16	4	0	4	5	One dead on 5th day—no infection. Good generalization in others
15	12 –9–36	12	5	18	8	1	7	5	Generalized infection in two, infection at inoculation points in five
16	1–18–37	11	5	35	73	20	53	6	All alive. Various degrees of infection
17	1–29–37	10	6	5	6	2	4	6	Very heavy generalized infection in all
18	3 –1–37	11	6	25	2	1	1	6	Extensive generalized infection
19	4 –5–37	9	6	29	4	1	3	7	Extensive generalized infection

*Membranes from three eggs used.

mune bird was mixed with an equal amount of virus suspension and incubated for about one hour. The control consisted of the same dose of virus with an equal quantity of broth.

In connection with the 18th generation, two eleven-day-old eggs received virus and the serum of a bird immunized against pigeon-pox. After incubation for six days, one egg showed three lesions at the inoculation points and the other showed a disc about 1 cm in diameter at the pole and generalized infection elsewhere. The two eggs inoculated with virus and the serum of a fowl-pox-immune bird showed generalized infection like the control.

The virus in a second test was from an 18th collateral generation. It had been passed through two generations of duck eggs, on March 31 and April 16, respectively. The virus was harvested from this second generation in duck eggs on April 22, and was used in the neutralization test on April 26. Here the results were somewhat suggestive of neutralization. Thus, two of the three twelve-day-old eggs inoculated with virus and pigeon-pox-immune serum survived six days of incubation. One embryo had died that day and showed no evidence of infection, while the second showed only two inoculation-point lesions. In the three eggs inoculated with virus and broth, there was generalized infection.

Virus from one of the control eggs in the above test, harvested on May 2, was used the next day in another test. The eggs were twelve days old and three of the four receiving virus and pigeon-pox-immune serum lived through six days of incubation. One showed two inoculation-point lesions and seven smaller lesions elsewhere. A second showed only three inoculation-point lesions. The third egg showed three inoculation-point lesions and six smaller secondary lesions. The four eggs that received virus and broth were a contrast in that the inoculation-point lesions were larger or confluent and generalization was much more extensive.

CULTIVATION IN ALIEN EGGS

Sixth-generation virus was inoculated into two eight-day-old pigeon eggs on June 8. One embryo was alive after incubation for seven days, but the membrane showed no evidence of infection. The other embryo died on the fifth day of incubation and, although there was no evidence of infection, an emulsion of the membrane inoculated into the feather follicles of a chicken on June 23 produced a suggestive "take." The lack of pigeon eggs prevented further trials.

Three twelve-day-old Muscovy duck eggs were each inoculated on March 31 (19th generation), with 0.8 cc of a suspension of 18th generation membrane. One embryo was dead by the next day and the other two died on the fourth day of incubation. The embryos were red, but the membranes showed no lesions and were not thickened. Nevertheless, a portion of one membrane was supended and inoculated into the follicles of a chicken. By the sixth day the follicles were greatly swollen. After holding in a frozen state for eleven days, the other portion of the membrane was suspended and inoculated into four ten-day-old hen eggs on April 16 (20th generation). One embryo died immediately, but the membranes of the three other eggs, opened after incubation for six days, showed very heavy infection. The curious fact to be noted here is that although the duck membranes used as inoculum showed no evidence of infection, they were rich in virus. Moreover, the duck embryos were apparently killed by the virus which has little lethal effect on the chick embryo. It should be added, however, that the incubation period of Muscovy duck eggs is 35 days instead of 28, as in Pekin ducks, so that inoculation on the twelfth day of incubation would be relative to inoculation of hen eggs on the seventh day.

The virus harvested from the above hen eggs (20th generation) on April 22, was passed through another generation of hen eggs on the 26th (21st generation), from which it was harvested on May 2. One of these membranes was used as inoculum for two thirteen-day-old Pekin duck eggs on May 3. After incubation for six days, both embryos were alive and a large disc was found at the inoculation pole of each egg.

Another membrane of the 20th generation, harvested on April 22, was held for 15 days and inoculated into eight twelve-day-old Pekin duck eggs on May 7. One embryo died the next day, and of the seven others, one died on the fifth day while six were alive on the sixth day when harvested. Every membrane showed extensive infection.

One of the membranes from the above (21st generation) was used to inoculate six fourteen-day-old Pekin duck eggs on May 17. The membrane had been held for four days in a frozen state. One embryo was dead on the third day of incubation and showed no evidence of infection. Another was dead on the fourth day and showed two inoculation-point lesions. The four remaining embryos were alive at this time and showed extensive lesions.

TITRATIONS

Several membranes from the fifth generation were dried *in vacuo* and graduated suspensions made by dilution containing from .0390625 mg to 20 mg of dried virus per cc of 50 per cent glycerin solution. Each dilution was inoculated into the feather follicles of chickens and a reading was made on the seventh day. All dilutions containing .625 mg or more of virus per cc gave "takes." That containing .3125 mg failed to infect, but the dilution containing .15625 mg gave slight infection.

Several membranes from the 16th generation were dried *in vacuo* at room temperature and held under refrigeration and tested. All dilutions gave "takes." The lowest tested contained .625 mg of virus per cc of diluent. These membranes were generally not extensively infected, nor were the "takes" very pronounced in the highest dilutions.

FIELD TESTS

Virus propagated in eggs was dried and used to vaccinate a total of 23,950 birds in 29 flocks under commercial conditions between June and October, 1936. The vaccine gave good "takes." That the pigeon virus does not give so high a degree of immunity as the fowl virus is well known. Nevertheless, of twelve non-infected flocks at the time of vaccination, containing 12,200 birds, only one was slightly affected with pox during the winter. The owner said it was of no consequence. One flock, containing 1,400 birds, was infected at the time of vaccination, but the owner was convinced that the outbreak was stopped after vaccination immunity had developed. No data are available on the remaining 16 flocks containing 10,350 birds.

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Veterinary Services in the Colonies

Since its recent formation the Advisory Committee on Oversea Services has been actively engaged in reviewing the conditions associated with the Colonial Veterinary Services.

From the results of this inquiry, a short report of which was made to the Council, N.V.M.A., at its recent meeting in Edinburgh, it is patent that, in certain ways at least, the service has been undergoing a period of retrogression, which has been steadily sapping its vitality and the enthusiasm of its members. The recruitment has fallen off and instances of established officers leaving the service prematurely, even at considerable personal sacrifice of pension rights, are not infrequent. Rightly or wrongly, these facts have suggested that the Colonial Veterinary Service receives no sympathetic consideration or understanding at the Colonial Office.—*The Veterinary Record.*

Can a Female Mule Conceive?

Mollie, said to be a dark bay mule, belonging to Wayne Mobley, Hartsville, Mich., is claimed to have given birth to a normal offspring. Opinions among veterinarians seem to be divided. What do you think?

Treatment of Infectious Sinusitis of Turkeys with Argyrol and Silver Nitrate*

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INTRODUCTION

Infectious sinusitis§ (swell head) of turkeys causes severe financial losses to growers each year. This disease, which has not received much attention from investigators, appears to be a different entity from the sinusitis observed by Hinshaw and Lloyd[1] to be associated with low vitamin-A intake. It also appears distinct from fowl coryza, which Beach and Schalm[2] succeeded in transmitting to turkeys.

The disease is characterized by nasal discharge, watery eyes, and swelling of the sinuses (chiefly infraorbital) on either or both sides of the face. The swollen sinuses usually are soft to the touch and contain a turbid, viscous, mucoid exudate which in the early stages may resemble egg albumin. In instances of long standing, or after improper treatment, the exudate may become caseated and firm. The mortality is usually low, but the morbidity varies from 10 to 90 per cent of the flock, and losses to the grower from birds of inferior or unsalable quality are often quite heavy.

The disease does not yield readily to the usual treatments of nasal irrigations and lancing. Tyzzer[3] reported the uneventful recovery of 15 turkeys with sinusitis that were treated by injecting 1 or 2 cc of fresh 15 per cent argyrol into the swollen sinuses after expulsion of the exudate by gentle pressure over the area. Madsen[4] reported 80 to 90 per cent recoveries after aspiration of the mucus and injection of 4 per cent silver nitrate into the affected sinuses. He also reported that argyrol, neo-silvol, tincture of metaphen, tincture of iodine, and calomel gave unsatisfactory results as compared with silver nitrate. For the past several years, we have advised aspiration of the mucus from, and injection into, the sinuses of about 1 cc of a fresh solution of 15 per cent argyrol. The results reported from this recommendation have been somewhat variable, although usually considered satisfactory.

This paper reports a series of field trials of argyrol and silver nitrate solutions as curative agents for the disease.

MATERIALS AND PROCEDURE

A 15 per cent aqueous solution of argyrol, or a 4 per cent aqueous solution of silver nitrate, were used in all trials. With two exceptions, both solutions were made up fresh the day before use. They were kept in brown glass serum bottles equipped with the regular rubber serum stoppers through which the contents could be withdrawn without danger of spilling. The turkeys were identified with leg bands.

The treatment was administered as follows: A 10- or 20-cc Luer-type glass syringe, fitted with a 12- or 15-gauge hypodermic needle, was used to aspirate the exudate from the sinuses. The attendant held the bird by grasping the skull just over the

FIG. 1. Infectious sinusitis. Aspirating the exudate from the infected sinus.

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§Infectious sinusitis is suggested as a name for this type of sinusitis to differentiate it from A-avitaminosis and fowl coryza.

ears with the left hand, while the left elbow and forearm lay across the underside of the right wing, binding the bird to the table. The legs were stretched and held in the right hand.

The sinus wall, when distended, was easily penetrated with the needle (fig. 1). If the wall was only slightly swollen and distended, the sinus was more easily penetrated when the beak was held open. The operator held the opened beak with the left hand and with the right hand he made a quick, short thrust of the needle through the wall of the infraorbital sinus as close to the cartilaginous structure of the beak as possible. As the exudate was withdrawn, it was found advisable to rotate the needle and syringe so as to prevent the mucous membrane from being drawn up into and clogging the lumen of the needle. Care was exercised not to damage the mucous membrane of the sinus with the point. After the exudate was aspirated, the syringe was released from the needle, which was left in the sinus wall. A 2-cc Luer-type glass syringe, filled with the solution to be injected, was then engaged with the needle, and 1 cc of the solution was injected into the sinus. Gentle massage was applied over the sinus, to distribute the solution, before the needle was withdrawn.

Two types of trials were conducted. The first consisted of controlled trials with six flocks, all the records of which were obtained by personally visiting the ranches. With the exception of flock 5, some of the affected birds were left untreated, as controls. However, the owner's enthusiasm over the results usually necessitated treatment of these birds at the time of the first examination. In the second type, for which four flocks were used, no controls were left, and all records taken subsequent to treatment were furnished by the owner.

CONTROLLED TRIALS

Flock 1: This flock consisted of 281 affected birds segregated from a flock of 1,000 5½-month-old Mammoth Bronze turkeys before and during the experiment. Sinusitis had been a serious problem for about two months, and various drugs had been administered. The treatment in use when the flock was taken for experimental purposes was lancing and swabbing the sinuses every two or three days with 2 per cent silver nitrate solution. The owner reported few recoveries.

At the first visit, 221 cases of sinusitis were presented for treatment. One hundred birds, of which 49 had been owner-treated previously, were treated with 15 per cent argyrol. One hundred twenty-one birds, of which 81 had been owner-treated previously, were held in the same pen, as untreated controls.

Examination of the birds one week after treatment revealed the recovery of 74 per cent of those treated, while but 14 per cent of the controls were found to have recovered. Three had escaped from each lot and could not be found for examination. Unfortunately, a large number of birds lost their bands and escaped from the segregation pen before the second examination two weeks after treatment. However, the examination, two weeks after treatment, of 70 treated birds and 68 untreated controls, showed that 81 per cent of the former and 18 per cent of the latter, had recovered. At the second examination, all birds still affected were treated with argyrol. Data on these were unobtainable, however, because most of them were sold before they could be examined.

In addition to the above, 60 new cases of sinusitis were separated from the main flock for treatment, of which 45 were treated with 15 per cent argyrol and 15 were left untreated, as controls. Examination one week later showed that 84 per cent of the treated, and but 18 per cent of the controls, had recovered. No further observations of this group were made.

Flock 2: From a flock of 1,400 Mammoth Bronze turkeys about five months old, a total of 228 affected birds were segregated and treated with a 2-month-old solution of 15 per cent argyrol. Ninety-three additional cases served as untreated controls. Examination one week later showed that 57 per cent of the treated, and but 9 per cent of the controls, had recovered. Those still affected were treated with 15 per cent

argyrol. Most of the birds were sold before a second examination could be made. The age of the argyrol is believed to be responsible, at least in part, for the lower percentage of recovery than was obtained in flock 1.

It is interesting to note that a large flock of chickens, kept on the farm and mingling to some extent with the turkeys, did not become infected.

Flock 3: In this flock of 31 Mammoth Bronze poults, eight weeks of age, 27 (or 87 per cent) were showing sinusitis in varying degrees. Four birds showed no symptoms and remained normal throughout the trial period of 72 days. The birds were all kept together in the same pen. Twenty-three were treated with a fresh 15 per cent solution of argyrol, while four were left untreated as controls.

Nineteen days later, twelve (or 52 per cent) of the treated birds, but none of the controls, had recovered. At this time, the eleven treated birds still affected were again treated, and the four controls were treated for the first time. Reëxamination, 24 days following this second treatment, revealed that 25 (or 92 per cent) of all birds treated had recovered. The two which had not were again treated and, when again examined 29 days later, were found to have recovered.

Only 40 feet away in outdoor brooders, chickens were being raised for broilers. At no time had there been any indication of coryza or sinusitis in these chickens.

Flock 4: From a flock of 1,600 5-month-old Mammoth Bronze turkeys, a total of 666 cases of sinusitis were available for treatment. Sinusitis had existed in the flock for about 45 days and no treatment had been attempted by the owner.

A total of 240 birds were treated with 15 per cent argyrol and 426 birds with 4 per cent silver nitrate. The first examination, eleven to 18 days after treatment, showed 72 per cent recoveries in the argyrol-treated group and 87 per cent recoveries in the silver nitrate-treated group. At this time, all birds that had not recovered were treated for the second time with the drugs used originally. One hundred and ninety-four birds that served as untreated controls for the first eleven days showed no recovery.

A second examination, 25 to 28 days after the first treatment, showed recovery of 86 per cent of 238 birds in the argyrol group and of 96 per cent of 416 birds in the silver nitrate group. Birds that had not recovered were treated in the same manner for the third time.

The third examination, 48 to 49 days after the first treatment, showed recovery of 94 per cent of 238 birds in the argyrol-treated group and 98 per cent of 286 birds in the silver nitrate-treated group. The birds that had not recovered were treated in the same manner for a fourth time.

A fourth examination, 60 days after the first treatment, showed recovery of 99 per cent of 171 birds in the argyrol-treated group and 100 per cent of 163 birds in the silver nitrate-treated group.

New cases were presented for treatment at the time of the first and second examinations. It was principally these that were examined during the third and fourth visits to the ranch. In other words, the third and fourth examination of the flock represented first and second examinations of the birds concerned. These were all recently developed cases and it was with them that the highest degree of treatment efficiency was obtained. These results show that recently-developed cases of sinusitis respond to treatment more readily than those of long standing.

Flock 5: In this trial there were 114 affected birds segregated from a flock of about 1,200. The disease had been present in the flock for about two months. The owner had been treating the worst cases of sinusitis by irrigation of the upper respiratory tract with a sodium hypochlorite solution, after manual expression of mucus from the sinuses. No appreciable number of recoveries had been obtained.

The birds were treated with 4 per cent silver nitrate solution. Several had partially-caseated exudate in the sinuses and, for the most part, such cases did not recover. The birds were examined at two and four weeks after treatment. At the

first examination, there were 108 birds left in pens, and 82 (75.92 per cent) of these had recovered. At the time of the second examination, only 98 were available and 85 (86.74 per cent) of these had recovered. The turkeys that were missing had escaped from the segregation pens into the main flock, and no attempt was made to reclaim them.

Flock 6: This flock consisted of 95 Narrangansett turkeys, twelve weeks old, among which sinusitis had been present for about a month. On October 14, six of the twelve affected birds, all bilateral, noncaseated cases then available, were treated with a 4 per cent silver nitrate solution that had been made up for at least two weeks. By November 8, all the treated cases had entirely recovered, but there was no improvement in the six untreated controls. These latter were segregated and left without treatment until January 15, 1938, when five were treated and one was left untreated. On January 26, the five treated birds were cured, whereas no improvement was noted in the control.

SUMMARY

As it was impossible to handle the treated flocks at regular intervals, a satisfactory summary of the results is difficult. An attempt, however, to tabulate the data of all flocks is made in table I. As stated previously, it was impossible to get the owners to leave untreated controls in the flock for more than one or two weeks. However, it is a well-known fact that spontaneous recovery from the disease occurs infrequently and, in any case, only after a prolonged period.

A factor that must be considered in comparing the effectiveness of argyrol and silver nitrate is the elapsed time between treatment and examination. In the first three field trials in which argyrol solution alone was used, the birds were examined one and two weeks after treatment. The results of these trials indicate a distinctly lower efficiency for argyrol than was given by silver nitrate in subsequent trials. However, in the subsequent trials in which a direct comparison of argyrol and silver nitrate was made and there was greater elapsed time between treatment and examination, the efficiency of the two drugs was more nearly equal.

UNCONTROLLED FIELD TRIALS

Besides the foregoing personally-conducted trials of treatment of sinusitis in turkeys, information has been obtained concerning four flocks in which the treatment was applied by the owners after they had been instructed with respect to procedure, methods, and records to be kept.

The first flock consisted of 2,800 Mammoth Bronze turkeys. Infectious sinusitis appeared when the flock was two months old and had been continuously present for about four months. Approximately 800 cases were removed for treatment. Many of them had been affected for at least a month. About half of the diseased birds were given 15 per cent argyrol, and the remainder a 4 per cent silver nitrate solution. Approximately 85 per cent of all cases were cured by the first treatment, 10 per cent more by the second, and 2 to 3 per cent by the third. The remainder did not recover. If treatment was administered early in the disease, no difference in the efficiency of the two drugs was noted. Silver nitrate seemed, however, more efficient for the advanced cases, especially if caseation had begun. The time for recovery varied from ten days to two weeks in early-treated cases and from five to six weeks in instances where caseation had started.

The second flock consisted of 15 or 20 turkeys, of which about half developed sinusitis. Treatment with 4 per cent silver nitrate produced complete recovery in about two weeks.

The third flock, consisting of 500 Mammoth Bronze turkeys, developed approximately 250 cases in two months, beginning when the birds were about four months old. Several methods of treatment, including lancing and swabbing the sinus with various chemicals had failed to effect cures. Treatment with 4 per cent silver nitrate brought about recovery of appoximately 90 per cent of the affected birds within six weeks.

The fourth flock consisted of 2,000 Mammoth Bronze turkeys in which infectious sinusitis first appeared about November 15, 1937, when the birds were five months old. Treatment with 4 per cent silver nitrate was begun on December 2. On December 15, a personal inspection was made. At that time, approximately 75 per cent of the treated birds had either recovered or were markedly improved. A later report from the owner stated that of 700 turkeys which were treated one or more times during about two months, approximately 500 made complete recoveries.

DISCUSSION

Infectious sinusitis readily responded to either 15 per cent argyrol or 4 per cent silver nitrate solution if the exudate could be successfully removed from the sinuses before the drugs were injected into them. The advanced cases responded less favorably and many had to be retreated several times before recovery took place. These results are in agreement with those reported by Tyzzer[3] concerning argyrol and Madsen[4] concerning silver nitrate. Our results with argyrol were better than those reported by Madsen. Dickinson and Beach,[5] using the same technic in one flock of chickens suffering from fowl coryza, found that 15 per cent argyrol was ineffective for this disease.

In tabulating the results, no differentiation was made between treatments for unilateral and bilateral sinusitis. In the first trials, treatment of unilateral cases consisted of injecting the affected sinus only and examination later often revealed that the opposite sinus had become affected. Subsequent to this observation, therefore, both sinuses were injected, irrespective of whether both were involved. This procedure reduced the number of cases that required a second treatment. The cases with bilateral sinus involvement responded to treatment as readily as the unilateral cases.

Always, a few minutes after treatment, the sinuses swelled again and sometimes to a greater size than before when the 4 per cent silver nitrate solution had been used. Unless the dosages were too large or the injections faultily made, however, no permanent injury to the tissue occurred. In two or three days, this swelling usually began to subside and, in over half of the birds, complete recovery occurred within ten to 14 days. A second treatment appears to be indicated for birds which do not show recovery or marked improvement within that time.

A possible reason for the relatively low efficiency of treatment with argyrol in flock 2 is that the solution used had been made up about two months prior to use. While it is not definitely known that a freshly-prepared solution would have been more effective, it is, nevertheless, thought advisable that only fresh solutions of argyrol be used in the treatment of sinusitis.

Although only slightly more efficient, the cheapness of silver nitrate in comparison with argyrol makes it preferable for use. A distinct disadvantage of silver nitrate, however, is its corrosive effect on both the instruments and the operator. When handling it, rubber or leather gloves should be worn.

CONCLUSIONS

1. Infectious sinusitis of turkeys can be successfully treated by injecting either 4 per cent silver nitrate or 15 per cent argyrol into the sinuses after the sinus exudate has been aspirated.

2. Silver nitrate is slightly more efficient than argyrol.

ACKNOWLEDGMENTS

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TABLE I—*Summary of treatment in flocks 1 to 6.*

METHOD OF TREATMENT	PERIOD OF EXAMINATION											
	1ST EXAMINATION 7 TO 19 DAYS AFTER 1ST TREATMENT			2ND EXAMINATION 14 TO 43 DAYS AFTER 1ST TREATMENT			3RD EXAMINATION 46 TO 72 DAYS AFTER 1ST TREATMENT			4TH EXAMINATION 60 DAYS AFTER 1ST TREATMENT		
	TOTAL NO.	RECOVERED†		TOTAL NO.	RECOVERED‡		TOTAL NO.	RECOVERED§		TOTAL NO.	RECOVERED¶	
		NO.	%		NO.	%		NO.	%		NO.	%
15% argyrol	696*	459	65.94	473	384	81.18	261	246	94.25	171	170	99.42
4% silver nitrate	534*	454	85.02	514	486	94.55	286	281	98.25	163	163	100.00
Untreated† controls	420	28	6.66	68	12	17.65	0	—	—	—	—	—

*All birds in this group had been treated once.
†Birds that had not recovered were treated in the same way a second time.
‡Birds that had not recovered were treated in the same way a third time.
§Birds that had not recovered were treated in the same way a fourth time.
¶Enthusiasm of owner prevented holding untreated controls after first examination.

Treatment for Tapeworms

A veterinary student who is employed in a small-animal hospital was the recipient of a cocker spaniel which was suffering from a skin affection. The dog was found to be infested with tapeworms. The usual method of withholding food and giving an anthelmintic failed to remove the parasites, so the student decided to try a new method.

He gave the dog a liberal feeding of veal, followed shortly with milk. When time enough had elapsed for the secretive fluids to begin digestion, the anthelmintic was given. The parasites were removed without any ill effects. The belief is that the ballooning of the intestines assists in the action of the anthelmintic. The student is following this treatment in all cases where there is difficulty in removing the entire tapeworm. The skin affection of his cocker spaniel disappeared entirely after the removal of the tapeworms.

The truly proud man knows neither superiors nor inferiors. The first he does not admit of; the last he does not concern himself about.—William Hazlitt.

Apologizing is a very desperate habit—one that is rarely cured. Apology is only egotism wrong side out. Nine times out of ten, the first thing a man's companion knows of his shortcoming is from his apology. It is mighty presumptuous on your part to suppose your small failures of so much consequence that you must talk about them.—Oliver Wendell Holmes, The Professor at the Breakfast Table, *Readers' Digest.*

Valuable Horseflesh

A consignment of 46 yearlings from Willis Sharpe Kilmer's Court Manor Stud, New Market, Va., went under the hammer in the sales paddock. They brought a total of $107,300, or an average of $2,333 per head. Top price for the sale was $12,000.

Why do you laugh? Change but the name and the story is told of yourself.—Horace.

Good men make me poor, bad ones make me rich.—Plautus.

Artificial Insemination of Ewes with Transported Semen*

By E. M. GILDOW, *Experiment Station Veterinarian, University of Idaho, and* C. E. TERRILL, *U. S. Bureau of Animal Industry, Moscow, Idaho.*

Research in artificial insemination during recent years has stimulated study and application of this valuable aid to breeding and genetic programs throughout the world. Undoubtedly, the application of the principle of artificial insemination for the utilization of outstanding proved sires will demand attention in the United States during the next few years.

One instance of pregnancies in sheep by artificial insemination with shipped semen has already been reported. Walton and Prawochenski[1] report the impregnation of two Polish ewes, one of which aborted, out of five inseminated with semen shipped from Cambridge, England, to Warsaw, Poland. The semen was 51 hours old and showed good motility when used.

Winters[2] reports successful impregnation of ewes with semen that had been kept in the laboratory five and six days. He also reports the successful shipment of semen from Saint Paul, Minnesota, to Grand Rapids, Minnesota. No statement was made concerning its use for artificial insemination.

During the fall of 1936, the Idaho Experiment Station at Moscow, Idaho, desired the use of a Lincoln ram in use at the U. S. Sheep Experiment Station at Dubois, Idaho. When it was found impossible to obtain the ram, an agreement was made to obtain semen from him for a preliminary study of artificial insemination. The problem involved the collection and shipment of the semen at Dubois, which was done by the junior author, and the determination of the ewes in heat and the insemination of such ewes, which was done by the senior author. Three ewes became pregnant and four normal, vigorous lambs were produced.

The semen was collected with a glass pipette from the vagina of a ewe not in heat. The semen was placed in small glass vials covered with mineral oil and packed in a vacuum bottle partly filled with cracked ice. It was then shipped by air and railroad express, a distance of approximately 700 miles. The time, between collection of the semen and time of insemination, ranged from 22 to 50.5 hours. Six shipments of semen were made. (See table II.)

In preparation for reception and use of the semen at the Idaho Experiment Station, Moscow, Idaho, 20 Lincoln ewes were segregated and the length of their estrous cycle as well as the duration of their heat periods, or estrum, was determined. (See table I.)

McKenzie and Phillips[3] have shown with Hampshires, Shropshires and Southdowns, that the average estrous cycle was 16.6 days in length and that the duration of estrum averaged 26.8 hours. No significant difference in age or breed was found for the length of the estrous cycle, but Hampshires had an appreciably longer estrum (30.7 hours), and yearlings showed a shorter estrum than older animals by 8.5 hours.

Observations on mature Lincoln ewes at the Idaho Experiment Station were made at twelve-hour intervals, using a teaser ram to detect ewes in heat. Only ewes that would stand for the ram were classified as in heat.

Table I shows that the average estrous cycle was 16.5 days, with a variation from 14 to 20 days in length. Of 54 estrous cycles measured, 46 (85 per cent) were between 15.5 and 17.5 days in length, four being under 15.5, and four being over 17.5 days. The average time in heat, that is, between the first and last evidence of standing for the ram, when checked at twelve-hour intervals, was 35 hours. The

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minimum time was twelve and the maximum 60 hours. Of 59 heat periods checked, 51 (86 per cent) were between 24 and 48 hours in length, five being under 24 hours and three over 48 hours. The actual length of estrum in these ewes was probably twelve hours longer than these data would indicate, since ewes were in heat a part of the twelve-hour intervals before the first and after the last time that they would stand for the ram. With information on one or more estrual periods for each ewe, the semen for artificial insemination was requested to arrive at a time when the largest number of ewes was expected to be in heat.

The use of gonadotrophic hormone might have eliminated the necessity of determining the length of the estrual cycle[4] but the data on length of estrum seemed essential. McKenzie and Phillips[3] have shown that ewes bred 14 hours or more after the beginning of estrum became pregnant much more regularly than those bred before that time. M. Kardimovitich,[5] as reported in a translation by V. Berliner, concludes that artificial insemination is most successful 22 hours after the onset of heat. The ewes were checked twice daily for estrum.

Ewes to be inseminated were closely confined in a crate that was equipped with a stanchion. The crate was elevated at the rear about 18 inches to facilitate the insemination technic. A pyrex vaginal speculum about 1 inch in diameter and 7 inches long, with the anterior end slightly tapered, was used to expose the *os uteri*. It was lubricated with neutral mineral oil. An electric head-lamp and a pen-type flashlight were used for illumination at different times. The flashlight was clipped to the inside of the speculum and was superior to the head-lamp. The semen was warmed for one-half hour in the vest pocket of the operator before being used. It was introduced undiluted in 0.2-cc portions with an ordinary straight 1-cc pipette graduated in tenths. The semen was deposited in the mouth of the servix at a depth of approximately ¼ to ½ inch.

All ewes that were in heat were inseminated at the time the semen arrived. Sixteen ewes were artificially inseminated one or more times. One ewe (R373) conceived the first time she was inseminated (semen A) and the other two (13 and 3210) the second time (semen F). (See table II.) The first ewe was inseminated 13 hours after she was detected in heat and the other two 16 hours afterward. The average length of estrum of these three ewes was 16, 24 and 40 hours, respectively. (See

Table I—*Length of estrous cycle and length of heat period in Lincoln ewes.*

Ewe	Ewe Age	Length in Days of Estrous Cycle				Length in Hours of Heat Periods			
		1st	2nd	3rd	Average	1st	2nd	3rd	Average
3	1	17.0	17.5	18.5	17.7	48	36	24	36
4	1	15.0	16.0	20.0	17.0	24	36	48	36
5	1	15.0	16.0	—	15.5	12	36	24	24
6	1	14.0	16.5	15.5	15.3	48	36	24	36
13	1	16.5	15.5	—	16.0	12	12	24	16
R184	4	19.5	14.5	15.5	16.2	24	12	24	20
R352	3	16.0	16.0	17.0	16.2	48	36	48	44
R373	2	16.0	—	—	16.0	24	24	—	24
2523	4	16.0	16.5	17.5	16.7	48	48	48	48
2525	4	15.5	16.5	17.5	16.5	48	48	36	44
2546	4	17.0	16.5	16.5	16.7	36	24	12	24
2661	3	16.5	18.0	17.5	17.3	36	48	24	36
2675	3	16.0	16.5	16.5	16.3	48	48	48	48
2736	3	16.5	17.0	16.5	16.7	36	48	36	40
2744	3	17.0	17.0	17.5	17.2	24	36	36	32
2747	3	16.0	17.0	15.5	16.2	24	48	24	32
2967	2	17.0	16.5	19.0	17.5	60	60	60	60
2982	2	15.5	15.5	—	15.5	36	24	36	32
3210	2	16.5	16.5	—	16.5	48	36	36	40
3212	2	16.0	16.5	17.5	16.7	36	24	24	28
Grand average		16.5				7			35

TABLE II— *Condition and age of transported semen at the time of insemination.*

SEMEN LOTS	A	B	C	D	E	F
Age in hours	27½	27	44½	22	43	44
Per cent motility	20 minus	0	60 plus	40 plus	20	80
Activity	Weak	None	Good	Good	Medium	Excellent
Texture	Poor	Good	Medium	Good	Good	Good
Ewes inseminated	7	0	5	4	7	4
Ewes conceived	1	0	0	0	0	2

table I.) Semen A, used on R373, was 27.5 hours old and semen F, used on 13 and 3210, was 44 hours old.

Ewe R373 produced a ram lamb; ewe 3210 produced twins, a ewe and a ram lamb, and ewe 13 produced a ram lamb. All lambs were normal and vigorous. Ewes 373 and 3210 had gestation periods of 146 days, while ewe 13 had one of 148 days.

Semen from the ram used, though normal in quantity, lacked considerably in quality. The concentration of spermatozoa was somewhat below normal. Two lots of semen were thick, lumpy and coagulated. The motility of the sperm at the time of collection was below that of other rams examined at the Dubois Sheep Experiment Station. The maximum time that the sperm from this ram was kept alive was about six days, with some lots living only two days. In contrast, sperm from other rams at the U. S. Sheep Experiment Station were kept alive at that station a maximum of 48 days, with a mean of 15 days.

SUMMARY

A preliminary investigation concerning the feasibility of shipping sheep semen for artificial insemination is reported. Twenty Lincoln ewes showed an average estrous cycle of 16.5 days and an average heat period of 34 hours. Sixteen ewes were artificially inseminated. The semen used was below average quality in motility and livability as compared with the semen of other rams at the U. S. Sheep Experiment Station. The semen was shipped from Dubois to Moscow, Idaho, a distance of approximately 700 miles. Three ewes became pregnant and produced one set of twins and two singles. The lambs were normal and vigorous. The semen used on one ewe that conceived was 27.5 hours old, and that used on the other two was 44 hours old. The shipment of semen for artificial insemination of sheep needs further study.

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New Principal

We are happy to extend felicitations to our confrerès on the appointment of Colonel Sir Arthur Oliver, C. B., C. M. G., F. R. C. V. S., as Principal of the Royal (Dick) Veterinary College, Edinburgh.

The July issue of *The Veterinary Journal*, from which source our information was obtained, states that because of Sir Oliver's outstanding qualities, no appointment will ever have given greater satisfaction to the members of the veterinary profession. Congratulations, Sir Oliver.

"Talking" Dog

Pat, a three-year-old Boston bull terrier, owned by Frank Elliott of Chicago, is reported to be able to carry on telephone conversations with an understandable vocabulary of about 14 words, including "yes," "no," "out," "eat," and "I want my mamma." This conversational ability is attributed to an enlarged larynx, according to the reporter.

What one does not need is dear at a penny.—Plutarch.

The Presence and Distribution of Hexamita sp. in Turkeys in California*

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Hexamita, a diplozoic flagellate, was first described by Dujardin[1] from stagnant water and from the intestines of amphibia. It also has been reported from fish (Schmidt,[2] Moore,[3] Davis[4]), from reptiles (Plimmer[5,6]), from Brazilian birds (da Cunha and Muniz[7,8]), from the duck (Kotlán[9]), from the pigeon (Nöller and Buttgereit[10]) and from rodents (Wenrich[11]). To our knowledge it has not previously been reported in turkeys.

We recently found this organism (as well as other protozoa in turkeys suffering from an eneritis similar to, and probably identical with, that described by Gierke and Hinshaw,[12] and Hinshaw.[13] They called attention to the presence of large numbers of *Trichomonas* in these cases, but no mention was made of other protozoa. In the acute stage of the disease, *Hexamita* is found in large numbers throughout the small intestine and in fewer number in the ceca. It is usually also found in the bursa of Fabricius, and in poults killed after they reached a comatose condition, it was found in a few instances in the abdominal cavity and in the liver. In the convalescent stages there seems to be a tendency toward a diminution in numbers in the duodenum and a persistence of large numbers in the lower jejunum, ileum and bursa of Fabricius. It has been found in the bursa in turkeys up to five months of age.

As in other forms of this order, the nucleus and other structures are duplicated so that the body has bilateral symmetry. There are six anterior and two long posterior flagella; in stained preparations the two axonemes stain deeply. The body is spindle-shaped and progresses in a fairly straight line with an exceedingly rapid, darting movement. This is in contrast to *Trichomonas* which, due to the presence of an undulating membrance, moves jerkily. The size and shape of the body and lack of a true cytostome serve to differentiate it from the roughly triangular *Chilomastix*. It varies in length from 6-12 μ (aver. 9μ), and in width from 2-5 μ (aver. 3 μ).

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Bossy Goes on "Spree"

When his usually placid, cud-chewing cow started jumping fences and cavorting about the pasture, a farmer called a veterinarian, who suggested the cow had been nibbling at marijuana and had become "tipsy." The wild dope weed was located in the pasture.

Transmission of Pasteurella Cuniculicida in Rabbits by Breeding*

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INTRODUCTION

Infectious diseases of lower animals known to be transmitted through the reproductive tract are comparatively few in number, and are confined, usually, to those conditions of the genital tract that have their primary foci in the genitalia. Infections ordinarily contracted by contact through the oral cavity, or through the nasal passages, are at times found to localize in the reproductive organs, but there are insufficient experimental data to evaluate the importance of such localized infections in the spread of the disease.

Bang's disease, primarily a disease of cattle, is known occasionally to become localized in the testicle of the male, and is thought to be disseminated in some cases from such a focus, although the more usual mode of infection is by the mouth. We have been unable, however, to find any previous report of pasteurellosis being transmitted through the reproductive organs of rabbits or other domestic animals. *Pasteurella* infections are prevalent in rabbits, as in most domestic animals, and it is possible that this mode of transmission has been disregarded because of the ease with which the disease is spread through direct contact.

During a recent study of *Pasteurella* infection in domestic rabbits, it was observed that when certain matings were made, the female often contracted an acute infection of hemorrhagic septicemia, while other females mated to the same buck would develop a purulent discharge from the reproductive tract that would result in sterility. The breeding record of one of these males revealed that some time previously he had been mated to a doe that was affected with an acute generalized case of hemorrhagic septicemia and died within 36 hours after the mating had been made. Subsequent breeding records of this male indicated that he might be transmitting the disease in some way, as a large percentage of the does mated to him died within six to twelve days later. As the disease was prevalent in the rabbitry at that time, other means of infection could not be precluded, but the suspected individual was retired from active service.

EXPERIMENTAL PROCEDURE

Experiment 1: A preliminary test was made to obtain information as to whether or not the disease was being disseminated from this male through contact or through breeding. Three cages were placed together so that the animals in the cages were separated by only a 1″ x 2″ piece of wire netting. The suspected buck was placed in the middle cage and two adult does in the cages on either side. Two more females were then mated to the suspect and, two days later, the same does were rebred to the same buck. After mating, these does were isolated in separate hutches. All animals used in the experiment were closely observed for a period of three weeks, but no symptoms or deleterious effects were observed.

Although the result of this experiment was negative, the suspected male was not returned to active service, but was retained in isolation for 13 months. Physical examinations of this animal during this interval revealed that the left testicle was slightly enlarged and was slowly increasing in size. The condition was diagnosed as an abscess of the testicle resulting from an infection with *Pasteurella cuniculicida.* The health and vigor of the animal seemed to be unimpaired.

Experiment 2: A second experiment was performed to test again the possibility of hemorrhagic septicemia being disseminated

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through breeding. Eleven adult does that had been isolated for three months and not bred during that time were used. These does were bred to the same buck used in the previous experiment that had given negative results.

The results of the matings, as shown in table I, were obtained from a daily observation of the animals as well as from bacteriological studies, and the date of observance of a purulent exudate from the reproductive tract or an acute septicemia of the exposed female was considered the date of effect. The females were carried to the hutch of the male and, immediately after mating, were returned to individual cages.

As a control group, four females were introduced into the hutch of the infected buck and were permitted to come into direct contact with him for about five minutes, mating being prevented, and then returned to individual cages.

Daily observations on the animals were supplemented by bacteriological study of those animals that died or showed a purulent discharge from the vagina. In the latter case, streak plates were made from vaginal swabs and positive infections were based on isolation and identification of *P. cuniculicida*. The identification of cultures was made by carbohydrate fermentation tests and cultural characteristics.

The results of this experiment, as shown in table I, were that two of the animals died two and three days, respectively, after being infected, and eight of the females developed clinical symptoms of the disease. One female that failed to develop symptoms of the disease from the first exposure was infected upon being exposed the second time, six weeks later.

There was a wide variation in the length of time required (2 to 42 days) to produce objective symptoms. In some cases there was inappetence, rough fur, and increased respiration, coincident with a purulent metritis and vaginitis. In other cases, however, the purulent exudate was the only symptom observed. The time required to produce this symptom is not necessarily indicative of the incubation period of the disease, as fatal cases of purulent metroperitonitis, without a discharge, caused from *P. cuniculicida*, have been observed.

TABLE I—*Results of experimental transmission of P. cuniculicida by breeding susceptible females to an infected male.*

DOE	BRED (1935)	EFFECT	DATE (1935)	RESULTS
306	10–9	None		Did not conceive
497	10–14	Dead	10–16	*P. cuniculicida* isolated. Generalized septicemia
309	10–16	Purulent discharge from reproductive tract	11–18	*P. cuniculicida* isolated from vaginal swab on streak plate
415	10–22	Purulent discharge from reproductive tract	11–1	*P. cuniculicida* isolated from vaginal swab and culture of exudate
429	10–28	Purulent discharge from reproductive tract	11–13	*P. cuniculicida* isolated from vaginal swab and culture of exudate
268	10–30	Purulent discharge from reproductive tract	11–16	*P. cuniculicida* isolated from vaginal swab
466	11–1	Purulent discharge from reproductive tract	11–13	*P. cuniculicida* isolated from vaginal swab
567	11–4	Purulent discharge from reproductive tract	11–13	*P. cuniculicida* isolated from vaginal swab and culture of exudate
684	11–7	Purulent discharge from reproductive tract	11–17	Cultures overgrown by contaminates
677	11–11	Purulent discharge from reproductive tract	11–18	*P. cuniculicida* isolated from vaginal swab
644	11–13	Dead	11–16	General septicemia. *P. cuniculicida* isolated
306	11–18*	Purulent discharge from reproductive tract	12–30	

*Rebred.
Note: Five controls (does 691, 430, 626, 620 and 586) were exposed on 11-2-35, 11-6-35, 11-10-35, 11-15-35 and 11-20-35, respectively, with negative results.

Experiment 3: In order to test the possibility of a buck acting as a mechanical carrier of the infection from an infected to a healthy doe, a healthy male was mated to a doe showing a purulent vaginal discharge from which *P. cuniculicida* was the predominant organism. Immediately afterward, this male was mated to a healthy doe. The following morning, the same male was mated to a second test female. To test the virulence of the organism in the exudate from the infected female, a small quantity was swabbed into the vagina of one test animal and into the conjunctiva of the second. The two does used to test the possibility of the organism being transmitted by the male, showed no symptoms of the disease. The doe inoculated intravaginally showed a purulent discharge on the fourth day and died on the sixth, while the animal inoculated in the eye showed no effects.

Uterine exudate from the fatal case was swabbed into the vagina of two new test animals, with the result that both animals died of generalized septicemia on the third and sixth days, respectively, after inoculation. Pure cultures of *P. cuniculicida* were isolated from the heart-blood, spleen, and uterine exudate of the fatal cases. The test animals that showed no effects from inoculation were sacrificed at the end of two months. The cultures made were negative for *P. cuniculicida*. Attempts were made to isolate *P. cuniculicida* from the test animals by means of vaginal swabs streaked on agar plates before they were inoculated. A group of 50 animals in addition to the control group were tested in this manner to determine if *P. cuniculicida* could be isolated from the uterus or vagina of healthy animals. In only one instance was it possible to isolate the organism from the reproductive tracts of animals showing no symptoms of the disease.

PATHOLOGY

At necropsy, the fatal cases of infection revealed the usual gross lesions of acute hemorrhagic septicemia, *i.e.*, petechial hemorrhages over subcutaneous and serous surfaces. Pure cultures of *P. cuniculicida* could be isolated from the heart-blood, liver and spleen without difficulty. Chronic cases of metritis due to *P. cuniculicida* revealed, upon autopsy, few gross lesions and those were confined to the uterus. The uterine horns consistently contained varying amounts of a thick, creamy exudate which was without pronounced odor. The endometrium was thickened, indurated and congested, and frequently the distention of the entire organ was very pronounced. Cases of this type were frequently encountered in which the uterus had become abcessed, finally rupturing into the peritoneal cavity or causing a fatal metroperitonitis.

To test the possibility of the uterus harboring an infection of *P. cuniculicida* without also infecting the vagina, half of the test group were slaughtered and the reproductive organs removed and cultured by searing and opening the horns of the uterus and vagina with sterile instruments, representative samples being taken from various sections. Results of this method corroborated results obtained by making streak plate cultures from vaginal swabs. In culturing the generative tract of large numbers of adult female rabbits, taken at random from the abattoir, for the presence of *P. cuniculicida*, it was found that the vaginal swab method of detecting the infection has limitations in that infections of long standing may cause abscesses within the uterus while the infecting organism may not be present in the vagina.

COMMENT

After the animals in the experiment were known to be infected with *P. cuniculicida*, cultures were made at intervals from vaginal swabs to determine if the infected females would continue to discharge the organism, and if such an infection would have a fatal termination. It was observed that the discharge of purulent exudate was periodic and that cultures made from vaginal swabs during a period when there was no evidence of exudate, were frequently negative for *P. cuniculicida*. The physical condition of the animals grew progressively worse, until the infection became fatal as a generalized septicemia, or formed extensive abscesses within the uterus resulting

in peritonitis. Some of the animals, sacrificed at the end of twelve months, were found to have retained the infection, but it was confined to the uterus.

The infected male lived for 14 months after experimental transmission of the disease, despite very extensive involvement of its reproductive organs. During the later stage of the disease, there was a continuous discharge of characteristic creamy, purulent exudate from the urethra. Upon necropsy, great enlargement of the left testicle was found, almost the entire glandular tissue being replaced by a large abscess. The organ was adherent to the *tunica vaginalis*, and the entire length of the spermatic cord was studded with small abscesses. The right testicle was not involved. The liver and spleen showed no gross changes. Cultures taken from the heart-blood were negative for *P. cuniculicida*, although direct smears and cultures taken from the abscess showed the organisms in abundance.

Transmission of *P. cuniculicida* through the reproductive organs is probably of minor importance in the transmission of the disease, as the usual means of spread, such as contact and inhalation, are more often responsible for epizoötics of this disease. The danger of such transmission by an infected male is apparent, however, as a carrier of this kind could infect a large number of females before the source of the infection could be detected. The immediate mortality might not be severe, as the experiments show that there is considerable variation in the susceptibility of individuals, but the most severe loss would occur from the large number of does rendered worthless for breeding purposes.

There are not sufficient experimental data from which to draw definite conclusions as to the importance of the male acting as a mechanical carrier of *P. cuniculicida* infection, but the results of experiment 3 would indicate that the disease is not easily transmitted in this way. Nevertheless, it would be extremely poor management to permit the mating of a doe showing any symptoms of a disease, although if an infected female should happen to be mated during a period when there was no visible discharge, the chance of transmitting the disease is remote, as it has been shown that at such times the infecting organism is not always present in the vagina.

Involvement of the testicles of the male is found only in exceptional cases, but careless practices, such as continued and repeated exposure of the male to infected does, are probably predisposing factors in establishing a focus of infection or abscess in the testicle. A doe showing the uterine form of *P. cuniculicida* infection is not presumptive evidence that the infection was transmitted by an infected male. Pregnant females, artificially infected, usually abort and cultures from the uterine mucosa in such cases show the presence of *P. cuniculicida*. Consequently, it is very probable that a generalized infection could become established in the uterus, under favorable conditions, regardless of the mode of infection.

Domestic rabbits frequently show large amounts of sediment of dietary origin in the urine, in addition to being susceptible to other types of uterine infection that are often mistaken for an infection with *P. cuniculicida*. It is, therefore, obvious that a diagnosis of pasteurelosis should be made in such cases only after an adequate study of the cultural and fermentative characteristics of the causative organism.

SUMMARY

Hemorrhagic septicemia of domestic rabbits infrequently becomes localized in the testicle of the male in the form of an abscess. Recognition of such infected animals is important, because females can regularly be infected from such a source.

The results obtained indicate that the strain of *Pasteurella cuniculicida* isolated is not readily transmitted by the healthy male from infected to non-infected females, although the wide variation in pathogenicity of different strains of *P. cuniculicida* as well as differences in individual susceptibility of test animals would probably affect the results obtained from the use of a different strain of the organism or from using a larger series of animals.

The Toxicity of Intestinal Filtrates from Lambs Dead of Overeating*

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A comparison of overeating[1,2] as described by this station, and enterotoxemia, as reported by Bennetts[3] in Western Australia, reveals that both diseases affect lambs in good condition and those on rich feed. Both are acute maladies and frequently result in death during the night without observed symptoms. Either may show convulsions and both are associated with rapid postmortem decomposition (pulpy kidney[4]).

Those signs which are regarded as characteristic of overeating, such as cerebral disturbance (throwing the head back, pushing against the fence), the presence of blotchy hemorrhages under the peritoneum and in the abdominal muscles and the diaphragm, the presence of quantities of plasma in the pericardial sac and, finally, the finding of from 2 to 6 per cent of sugar in the urine, are not described by the Australian investigators.

In spite of these discrepancies, it seemed that the toxicity of intestinal filtrates taken from lambs dead of overeating should be determined. Accordingly, the work was started in 1932 and has been continued to the present time.[5] This paper is a brief summary of one phase of the broader problem which will be reported in detail at a later date.

MATERIALS AND METHODS

The small intestines were selected from the lambs as soon as possible after death, stripped of their contents, an equal amount of 0.85 per cent salt solution added and the material filtered through several layers of gauze. This was then centrifuged at about 2,500 r.p.m. for an hour, the supernatant fluid drawn off and passed through a clay filter. After sterility had been checked, it was inoculated intramuscularly into the thighs of guinea pigs, usually in 4-cc doses, 2 cc on each side. Bekefeld and Mandler filters were used during the first two seasons (1932-34). In 1934-35, the material was passed through Chamberland and then through Seitz filters.

Beginning in 1935, the supernatant fluid after centrifugation was filtered three times through filter paper, first through four, eight and then twelve layers moistened with water. It was then injected intravenously into rabbits in from 0.5-cc to 1.2-cc amounts, according to size and, if not toxic, was discarded. If toxic, it was sterilized by passage through a Seitz filter.

During the first two years, guinea pigs were injected intramuscularly. During the rest of the time, rabbits were inoculated intravenously. Mice were used at various times but only for check tests.

RESULTS WITH LABORATORY ANIMALS

Reference to table I will show that, over a period of six years, 343 filtrates were thus tested, 256 being from lambs dead of overeating and 87 from those dead of other diseases. Of the 256 from the cases of overeating, 115 (45 per cent) were toxic either for guinea pigs or rabbits, while of the 87 dead from other diseases, only two were injurious to these laboratory animals. One of these two was taken from a lamb dead of coccidiosis and one was from an animal dead of a ruptured bladder. Looking back over the records of these cases in the light of the negative results of subsequent years, there can be little doubt of their authenticity. Both were diseases that were unlikely to be mistaken and in both instances so much work was done with the filtrates that there can be no question of the toxicity.

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†Due to frequent changes in the staff, the present report is the composite labor of a number of workers. While the senior author has been in touch with the problem throughout, the following should be given credit for the details: Floyd Gross, 1932-34; B. R. McCrory, 1934-35; A. H. Groth, 1935-36; Frank Thorp, Jr., and A. W. Deem, 1936-38.

TABLE I—*Toxicity for laboratory animals of intestinal contents from lambs.**

SEASON	OVERHEATING				OTHER DISEASES			
	TESTED	TOXIC		NON-TOXIC	TESTED	TOXIC	NON-TOXIC	TOTAL TESTED
		NO.	%					
1932–33	24	9	38	15	0	0	0	24
1933–34	24	4	17	20	6	1	5	30
1934–35	27	5	19	22	14	1	13	41
1935–36	51	21	41	30	5	0	5	56
1936–37	76	38	50	38	20	0	20	96
1937–38	54	38	70	16	42	0	42	96
Total	256	115	39	141	87	2	85	343

*During the first two years, guinea pigs were injected intramuscularly with 4 cc. During the remaining years, rabbits were given from 0.5 cc to 1.2 cc intravenously, according to weight.

RESULTS WITH SHEEP

During the first season (1932-33) eight of the nine toxic filtrates were injected into the axillae of sheep in 25-cc doses equally divided between the two sides. Six of the injected animals died within 24 hours. The two others survived after showing swelling and lameness. The lesions of the dead sheep consisted of large accumulations of sterile clear plasma, non-gaseous and non-hemorrhagic, in some instances extending downward to the knee, forward to the throat and backward to the lower flank. The remaining filtrate was not used because the amount was too small to justify injecting it.

In 1933-34, five filtrates were found toxic for guinea pigs and, of these, four were injected into the axillae of sheep in 25-cc doses. Three of the sheep died with lesions as in the previous year and one survived after being desperately ill for 24 hours. One of these filtrates which killed a sheep was from a lamb dead of coccidiosis. In two instances, 10-cc doses failed to kill sheep where 25-cc doses had done so.

In 1935-36, only two filtrates were tested on sheep. One of these killed sheep in doses of 25, 12 and 5 cc and the other failed to kill in 15-cc dosage.

In 1937-38, six filtrates were tested on sheep. One of them killed in 30-cc dosage but failed to kill in amounts of 12 and 5 cc, and another in 65 cc. Two others produced toxic symptoms after the administration of 50 cc but did not destroy.

Special attention was given to the urine in all cases, but in most of them the bladder was empty. In three instances sugar was present. In one of the 1932-33 series, 0.42 per cent sugar was present and in another just a trace. One of the 1933-34 animals showed 1.43 per cent sugar. Urine was obtained from the two sheep in 1937-38 and, while both showed albumen, neither gave a test for sugar.

TABLE II—*Toxicity of intestinal filtrate for sheep.*

SEASON	INJECTED	KILLED	SURVIVED
1932–33	8	6	2
1933–34	4	3	1
1935–36	2	1	1
1937–38	6	2	4
Totals	20	12	8

Only one animal showed the blotchy hemorrhages considered typical of overeating, that being one of the 1937-38 lot.

THE NATURE OF THE TOXIN

In the earlier years, each toxic filtrate was heated in a water bath of 60° C. for 30 minutes and then injected into guinea pigs in the same dose that had proved fatal. In every instance the guinea pig survived, indicating that the heating had destroyed the

toxin. In the later years, routine heating was frequently neglected, but in those cases where the effect of heating was tested, the results were the same.

In 1934-35, the five toxic filtrates were incubated with anti-toxins kindly furnished by the Wellcome Laboratories, of London, England, to determine the neutralizing effect of these serums. They were labeled *"B. welchii," "B. paludis," "B. ovitoxicus"* and "lamb dysentery." While there was some variation in the tests conducted, in general 0.5 cc of the filtrate was incubated at 370° C. with 0.5 cc of the antiserum for one hour and the whole was then injected intravenously into a rabbit. In every trial the *B. ovitoxicus* (Wilsdons type D) and lamb dysentery (Wilsdons type B) neutralized the filtrate, while the *B. welchii* (Wilsdons type A) and *B. paludis* (Wilsdons type C failed to do so.

These filtrates failed to cause symptoms in rabbits when given by the mouth in 10-cc doses.

In 1935-36, eight of the filtrates were tested only against antiserums for *B. welchii, P. paludis* and *B. ovitoxicus*, the lamb dysentery serum having become contaminated in the meantime. All eight were completely neutralized by *B. ovitoxicus* serum but remained toxic in the presence of the two others.

DISCUSSION

Bennetts found 14 toxic filtrates out of 16 examined (88 per cent), which was higher than in any season reported here. In the earlier years, however, the manipulation necessary to obtain sterility may have been responsible for some of the weaker filtrates losing their toxicity. In some years, also, the filtrates were held a considerable time before being tested, owing to a shortage of appropriate test animals. In the 1937-38 season, when most of these difficulties were overcome, the percentage of toxicity ran 70, thus more nearly approaching Bennetts' figures.

Bennetts found, as in this study, that his filtrates were neutralized by both the lamb dysentery and ovitoxicus antiserums and were not rendered harmless by antitoxins made from *B. welchii* and *B. paludis*.

If it can be assumed that overeating is due to the multiplication of *B. ovitoxicus* (*Cl. welchii* type D) and the elaboration of its toxin in the intestinal tract, then it seems that a reasonable explanation for the development of the disease is that the consumption of a large amount of concentrate tends to develop conditions favorable to the growth of that organism. As a practical procedure, the disease has been controlled during the past 30 years by reducing the grain ration and increasing the roughage content of the diet.

While the toxin is undoubtedly injurious to laboratory animals as well as to sheep, when injected parenterally, the authors have so far been unable to confirm Bennetts' finding and demonstrate its toxicity when given by the mouth. It can therefore only be assumed that it so injures the epithelium of the small intestine that it is absorbed into the circulation.

SUMMARY

Forty-five per cent of intestinal filtrates from 256 lambs dead of overeating were shown to be toxic for laboratory animals.

Of 87 similar filtrates from lambs dead of other diseases, only two were toxic, one from a lamb dead of coccidiosis and one from a case of ruptured bladder.

Twelve out of 20 of the filtrates proved fatal to sheep when injected in amounts varying from 5 to 50 cc.

Heating the filtrates to 60° C. for 30 minutes rendered them harmless.

The toxin was neutralized by antiserums made from the lamb dysentery bacillus and from *B. ovitoxicus* but not by those made from *Cl. welchii* or *B. paludis*.

While overeating seems to differ from enterotoxemia on the basis of symptoms and lesions, the intestinal contents of lambs dead of the two diseases contain toxic substances that seem to be identical.

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Lechuguilla (Agave Lecheguilla) Poisoning in Sheep and Goats*

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Grazing on the leaves of lechuguilla, by sheep and goats, is frequently associated with serious losses in the Southwest. This form of poisoning is generally referred to as "lechuguillaed," "goat fever," or "swellhead," and is characterized by icterus and a degeneration of the liver and kidney parenchyma. In addition to the constant lesions, an edematous swelling of the face and ears similar to that of other photodynamic diseases is frequently, but not constantly, observed. In previous publications,[1] it has been shown that the complete clinical picture of lechuguilla poisoning represent the combined action of two toxic principles, one a hepato-nephro-toxin which has been identified as a saponin, the other a photodynamic agent. A review of the literature on photodynamic diseases has also been presented[2] and is, therefore, cmitted at this time. The present publication is confined to a report of the clinical and experimental study of the disease in sheep and goats.

OCCURRENCE

Drouth and unfavorable range conditions being the chief contributing factors to the occurrence of this disease, the most serious losses are generally sustained during the spring of the year, following a period of drouth. However, sporadic outbreaks may, and frequently do, occur during any or all seasons of the year. The morbidity varies from 5 to 30 per cent, with the mortality approaching the same percentage, since under ordinary range management the affected animals are left to shift for themselves and, as a result, recovery is the exception rather than the rule. Outbreaks are readily checked by removal of the flock from infested to non-infested pastures, but in western Texas this method is available to but a small percentage of the ranchers. Consequently, the only available recourse is to move to another locality or to hope for improved climatic conditions and the appearance of a more palatable forage.

FIG. 1. *Agave lecheguilla* plant.

SYMPTOMS

A listless attitude and inability to follow the remainder of the flock are the first evidence of intoxication. A yellow, inspissated exeretion adheres to the internal canthus of the eye and may mat the hair or wool for some distance below this point. A nasal discharge of a yellow, tenacious character is generally observed. Inappetence appears at an early stage and becomes more pronounced as the disease approaches a fatal termination. Icterus of the sclera, skin and visible mucous membranes is pronounced. The urine is clear amber but occasionally port wine in color. There are a progressive weakness and emaciation with a short period of coma preceding death. Pruritus is not so prominent a manifestation in this disease as it is in some of the other photodynamic diseases.

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‡In coöperation with the U. S. Department of Agriculture, Bureau of Animal Industry.

PATHOLOGY

Upon autopsy, a generalized icterus and retrograde changes in the liver and kidneys are constantly observed. The liver is brownish-yellow in color and from its gross appearance it is evident that fatty changes have occurred. Incision, followed by the application of pressure along the edge of the cut surface, results in the expression of numerous plugs of thickened bile from the severed bile-ducts. The bile may be normal in appearance, but it is not uncommon to find the content of the gall-bladder doughy in consistence. The kidneys are greenish-black in color and may be swollen, sometimes to as much as twice their normal size. The greenish-black background, after removal of the capsule, is found to be studded with numerous gray foci slightly smaller than the head of a pin. Upon incision, urine drips from the cut surface and the tubules may be sufficiently distended to be identified with the unaided eye.

In addition to the constant lesions, an edematous swelling of the face and ears, which may extend down into the intermandibular space, is of frequent occurrence during the late spring, summer and early fall months. This condition is reported as occurring under range conditions during the winter months, but has never been observed by the author during this season. The edematous swelling, or the reaction to sunlight exposure, is of more frequent occurrence at the beginning than it is towards the termination of an outbreak. In mild or moderate reactions, the edema is quickly reabsorbed. In severe reactions, rupture of the skin, with escape of the edematous fluid, is frequently observed. Reabsorption or drainage of the edema is accompanied by necrosis of the skin covering the involved areas. Mummification, followed by sloughing of a part of one or both ears, may be observed in the occasional case which terminates in recovery. A less frequent manifestation is a wrinkled, rigid and opaque cornea.

The microscopic pathology, as in the case of the gross, is dependent upon the etiology. Evidence of photodynamic action is to be found in the skin and adjacent

FIG. 2. Sheep 54A sensitized with the hydrolized alcohol extract of lechuguilla.

connective tissue, whereas the liver and kidneys exhibit evidence of the toxic action of the saponin. The primary lesion of photodynamic action is to be found in the deeper layers of the corium and adjacent connective tissue and consist of an intracellular edema of the capillary endothelium, accompanied by an edematous infiltration of the surrounding tissue. Later, leukocytic and reticuloendothelial accumulations, together with regeneration of the endothe-

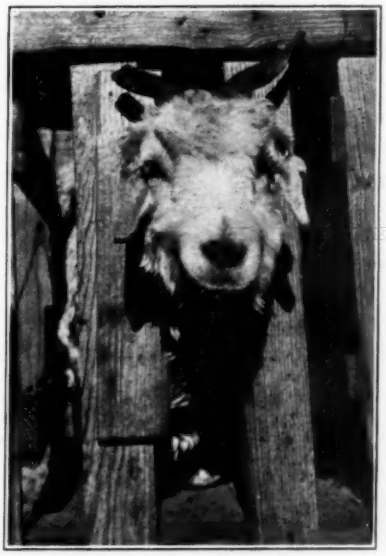

FIG. 3. Goat 99 (12-12-36) sensitized by feeding the leaves of lechuguilla.

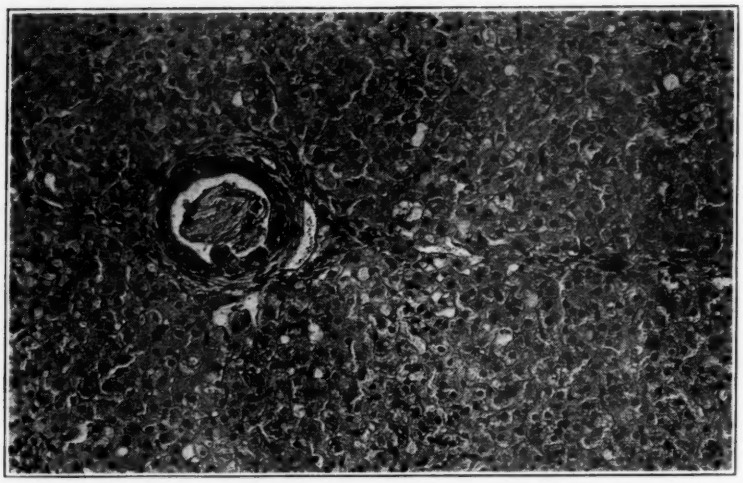

FIG. 4. A photomicrograph of the liver from a case of lechuguilla poisoning, showing some degenerative changes and a cholesterol case in the bile-duct.

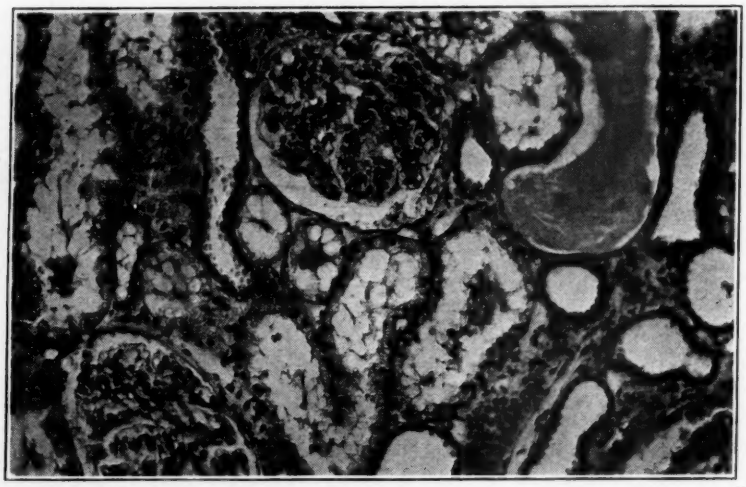

FIG. 5. A photograph of the kidney from a case of lechuguilla poisoning, showing a degeneration of tubular and glomerular epithelium, distention of the tubules and case formation.

lium, give the walls of the capillaries a striking rich, cellular appearance. This reaction is much more pronounced in the deeper layers of the corium than it is in the more superficial layers.

There is a moderate infiltration of wandering cells into the edematous areas which becomes predominantly polymorphonuclear in type as necrosis of the skin develops. Discrete hemorrhages are frequently observed. Necrosis of the skin, accompanied by an inflammatory exudate, occurs as early as the third day and becomes quite extensive by the fifth day. The epithelium of the sebaceous glands, especially of the skin of the ears, shows marked necrosis with liberation of the lipoid material and a polymorphonuclear leukocytic infiltration. In the skin of the ears, the reaction to light exposure is confined to the outer surface, the inner surface showing little or no change.

There is a marked degenerative fatty infiltration of the liver parenchyma, local necrosis, and deposits of bile pigment in the degenerated cells. Cholesterol casts, which are surrounded by an amorphous, brownish-stained material, occlude many of the bile-ducts. The kidney parenchyma shows the same fatty changes, but the necrobiotic changes are more pronounced than they are in the liver. Numerous large casts, composed of degenerated cells and precipitated albumin, occlude the tubules, resulting in an enormous tubular distention above the occlusion. The retrograde changes occur in both tubular and glomerular epithelium.

EXPERIMENTAL REPRODUCTION OF LECHUGUILLA POISONING

An exclusive ration of lechuguilla: In a series of three experiments, twelve Angora and four brown or brown and black goats were forced to eat the leaves of lechuguilla by withholding all other food. Thirteen of the goats were fed in open pens and, therefore, exposed to the available sunlight for the duration of the experiments. The remaining three were maintained in a building in diffuse light for like periods. Three of the animals were fed during the month of December, six during February and March, and seven during May. Marked evidence of intoxication developed after feeding periods varying from nine to 25 days, the average consumption being 8.8 pounds during an average feeding period of 18 days.

The mortality was 75 per cent. Without exception, these animals developed the usual icterus, liver and kidney lesions, but showed no evidence of a photosensitization. The absence of photosensitization in these experiments could not be attributed to insufficient sunlight exposure, as clear weather prevailed for the duration of the three experiments. Since the pathology which was produced in these experiments was the same, irrespective of light conditions or the color of the animals, it is evident that this part of the clinical picture of lechuguilla poisoning is not a photosensitization.

LECHUGUILLA PLUS ALFALFA HAY AD LIBITUM

In the previous experiments, starvation was an uncontrolled factor which probably contributed to the mortality. This factor was therefore eliminated in the following experiments. In another series of experiments, weighed amounts of lechuguilla were force-fed and alfalfa hay allowed *ad libitum* to nine goats and eleven sheep. The results of the experiments are summarized in table I.

In the first experiment, which was conducted during August, five animals received daily exposure to sunlight, with one animal maintained in a building as a control. Three of the animals (sheep 55, 58 and goat 93), which were exposed to sunlight, reacted with the complete clinical picture of lechuguilla poisoning, the lesions of photosensitization appearing on the ninth, twelfth and 16th day, respectively. The control (sheep 30) developed the constant lesions of the disease (icterus, hepatitis and nephritis), but no edematous swelling of the head.

No toxic effects were observed as a result of feeding the plant to the two remaining animals (sheep 37 and goat 72). Since 1-pound doses for 24 consecutive days had no toxic effects, it was assumed that a re-

sistance to both toxic principles had been developed in these animals by a preliminary feeding period of seven days, during which time they were fed ¼ and ½ pound per day, respectively. If such were the case, the resistance to the hepato-nephro-toxin was of short duration, as 1-pound doses for six and seven days, respectively, were later found to be sufficient to produce this part of the disease.

During September and October, three goats and two sheep were fed with this plant and exposed to sunlight. Marked evidence of the toxic action of the saponin was observed in these cases, as well as in the controls, but no evidence of a photosensitization. Failure to produce photosensitization in these animals was first attributed to insufficient light energy, as cloudy weather prevailed during the feeding period in September and October. Although clear weather prevailed, a similar reaction could not be obtained in known sensitized rats. However, subsequent experimentation proved that the lack of sufficient light energy was not the true explanation.

Goats 96 and 97 and sheep 63 and 64 were placed on experiment on November 9 with the object of reproducing a resistance to the toxic saponin similar to that which had been produced in August. These animals were maintained in a shed having an open southern exposure, with no special attention being given to direct sunlight exposure. By November 21, it was obvious that no resistance to the toxic saponin had developed and the feeding of lechuguilla was discontinued. The weather conditions between the ninth and 21st are probably significant in considering the results which were later obtained.

November 11, 12 and 13 were clear, bright days, but from the 14th to the 21st, cloudy weather prevailed with the addition of fog on the 19th to 21st inclusive, thus excluding the possibility of a sunlight exposure on these three days. On the 22nd, a bright sunlight exposure occurred for about three hours during the middle of the day, although the animals could have avoided the exposure had they so desired. The two goats exhibited a marked, edematous swelling of the face on the 23rd, but the sheep were not affected. The 23rd was again cloudy. On the 24th, sheep 64 was exposed to bright sunlight from 8:00 a. m. to 5:00 p. m. Sheep 63 was maintained in a building and not exposed.

By the termination of the exposure, sheep 64 was showing a moderate swelling about the face and ears, a reaction which was intensified by a similar exposure on the following day. Until December 1, sheep 63 remained free of a similar reaction. It was therefore exposed to sunlight on December 1 to 3 inclusive, but at no time did it exhibit evidence of a photosensitization. Goats 98 and 99 and sheep 65 and 66 were fed lechuguilla in diffuse light from November 24 to December 9. On the latter date, goat 99 and sheep 66 were exposed to bright sunlight from 10:00 a. m. to 3:00 p. m. Both animals showed a moderate swelling of the face and ears on the following day. This reaction was intensified and accompanied by marked pruritus during a second light exposure on December 10. The action of the hepato-nephro-toxin, but no evidence of a photosensitization, was observed in the two animals which were not exposed.

THE FEEDING OF EXTRACTS OF LECHUGUILLA

The ground green leaves of lechuguilla were covered with distilled water and allowed to stand for ten to 14 hours at room temperature. This extract was filtered and brought to the desired concentration by boiling. The concentrated extract was fed to six goats and two sheep on the basis of dry solids. The size of the daily dose varied from 200 to 275 grams. The number of doses varied from one to six. The animals were maintained in open pens and, therefore, exposed to all available sunlight for the duration of the experiments. Two of the animals were fed in December, three in January, and three in August. One animal died ten hours after receiving a single dose of the extract. Death in this case was attributed to an acute action of the toxic saponin, since no evidence of an acute photosensitization was observed. The seven remaining animals showed marked evidence of the action of the toxic saponin, but with one exception, no evidence of a photosensi-

tization. The one exception showed a moderate swelling of the ears, but no swelling of the face. A mortality of seven of the eight animals furnished ample proof of the toxic action of the extract, but the toxicity was confined to that of the saponin, with a doubtful photosensitization in the one case.

The dried, ground leaves of lechuguilla were covered with 96 per cent ethyl alcohol and extracted at boiling temperature for two to three hours. The hot alcohol was decanted and the extraction repeated two times. The three extractions were combined, filtered while hot and again upon cooling to room temperature. The alcohol was evaporated, the residue taken up in distilled water and administered as an aqueous solution on the basis of dry solids. This extract was fed to one sheep at the rate of 46 grams a day for two consecutive days. Death occurred about ten hours after receiving the second dose. This animal was exposed to direct sunlight, but as there were no symptoms of an acute photosensitization, it was assumed that death was due to an acute action of the toxic saponin.

From the work with laboratory animals, which has been previously reported, it was found that the toxic saponin could be hydrolyzed with hydrochloric acid, without destruction of the photodynamic agent. A portion of extract was thus treated, neutralized with sodium hydroxide, and fed to one sheep. This animal was fed 50 grams a day for four consecutive days and exposed to sunlight each day. Photosensitization was well marked in this animal 72 hours after receiving the first dose of the hydrolyzed extract. A slight icterus developed on the fifth day, thus indicating that hydrolysis of the toxic saponin was incomplete. The extract was, therefore, hydrolyzed an additional four hours, neutralized and fed to three sheep at the rate of 46 grams a day for three consecutive days. Two of the sheep were maintained in diffuse light. The third animal was exposed to direct sunlight. It developed marked lesions of photodynamic action on the fifth day after receiving the first dose. No toxic action was observed in the two animals which were maintained in diffuse light. Since there were no clinical manifestations of the action of the hepato-nephro-toxin in these three animals, it was evident that this toxic principle had been hydrolyzed without destruction of the photodynamic agent.

SYMPTOMS AND PATHOLOGY OF EXPERIMENTAL CASES

The manifestations of lechuguilla poisoning under experimental conditions were practically the same as those which were observed in natural outbreaks of the disease. Pruritus during the development of the photodynamic reaction was a prominent manifestation in many of the experimental animals, thus indicating that this symptom is probably more prevalent under range conditions than is generally observed. An acute photosensitization similar to that observed in some of the other photodynamic diseases was not observed. As a rule, the swellings of the face and ears attained a maximum degree by the end of the second day. Reabsorption of the edema began on the third and was generally complete by the end of the fourth or fifth day. Rupture of the skin covering the edematous areas was not observed. Temperatures of 106 to 107° F. were noted during the development of the photodynamic reaction, but returned to normal with the termination of this reaction. Subnormal temperatures were an indication of a fatal termination. High temperatures in lechuguilla poisoning without photosensitization have never been observed.

The most significant blood changes consisted of a leukocytosis with an increase in the percentage of polymorphonuclear leukocytes and a marked increase of the non-protein nitrogen content of the blood. Evidence of kidney degeneration was reflected by an increased non-protein nitrogen content of the blood as early as the eighth day and, by the twelfth day, values of over 100 mg per 100 cc of blood were of common occurrence. There was a marked decrease in the erythrocytic content of the blood in some cases but not in others. The blood studies of two representative animals are listed in table II. These studies were con-

TABLE I—*The toxic action of lechuguilla, alfalfa hay ad libitum.*

ANIMAL	WEIGHT (LBS.)	DAILY DOSE (LBS.)	DATES—INCLUSIVE (1936)	RESULTS
		EXPOSED TO DIRECT SUNLIGHT		
G72	85	.5	8–10— 8–16	No ill effects
		1.0	8–17— 9–9	No ill effects
S37	65	.25	8–10— 8–16	No ill effects
		1.0	8–17— 9–9	No ill effects
G93	50	.25	8–10— 8–16	No ill effects
		.5	8–17— 8–25	Ps. 8-25, icterus, died 9-4
S58	70	.5	8–10— 8–21	Ps. 8-21, icterus, killed 8-24
S55	75	1.0	8–17— 8–25	Ps. 8-25, icterus, killed 8-29
G92	60	1.0	9–9 — 9–19	No Ps., icterus, recovered
S37	62	1.0	10–5 —10–10	No Ps., icterus, recovered
S47	50	1.0	10–5 —10–10	No Ps., icterus, died 10-20
G94	90	1.0	10–5 —10–17	No Ps., icterus, died 10-22
G96	60	.25	11–9 —11–13	No ill effects
		.5	11–14—11–16	Loss of appetite
		1.0	11–17—11–21	Ps. 11-22, icterus, died 11-24
G97	80	.25	11–9 —11–13	No ill effects
		.5	11–14—11–16	Loss of appetite
		1.0	11–17—11–21	Ps. 11-22, icterus, died 11-26
S64	70	.25	11–9 —11–14	No ill effects
		.5	11–15—11–16	Slight loss of appetite
		1.0	11–17—11–21	Ps. 11-24, icterus, recovered
S63*	65	.25	11–9 —11–14	No ill effects
		.5	11–15—11–16	No ill effects
		1.0	11–17—11–21	No Ps., icterus, recovered
		FED IN DIFFUSE LIGHT		
S30	72	.5	8–10— 8–25	No Ps., icterus, recovered
S54	90	.5	9–9 —10–3	No Ps., icterus, recovered
G84	90	1.0	10–5 —10–20	No Ps., icterus, died 10-26
S62	120	1.0	10–5 —10–20	No Ps., icterus, died 11-1
G98	75	.25	11–24—11–28	No ill effects
		.5	11–29—12–1	No ill effects
		1.0	12–2 —12–6	No Ps., icterus, recovered
G99†	80	.25	11–24—11–28	No ill effects
		.5	11–29—12–1	No ill effects
		1.0	12–2 —12–6	Ps. 12-9, icterus, killed 12-24
S65	60	.25	11–25—11–30	No ill effects
		.5	12–1 —12–2	No ill effects
		1.0	12–3 —12–6	No Ps., icterus, recovered
S66†	70	.25	11–25—11–30	No ill effects
		.5	12–1 —12–2	No ill effects
		1.0	12–3 —12–6	Ps. 12-9, icterus, died 12-13

*Not exposed to sunlight from 11-22 to 11-30, but exposed on 12-1.
†Exposed to bright sunlight 12-9, 10.
Ps. = Photosensitization.
No Ps. = No photosensitization.

ducted from the beginning to the termination of the feeding period.

DISCUSSION

Results of experimental work with laboratory animals showed that resistance to photodynamic action developed as a result of insufficient sunlight exposure and upon recovery from the disease. The absence of this part of the clinical picture in the natural occurrence of lechuguilla poisoning may, therefore, be due to two factors; first, reabsorption of the edema or recovery from this part of the disease and second, the development of resistance without injury to the blood-vessels. The relatively rare occurrence of this reaction under range conditions during the winter months sug-

TABLE II—*Blood studies of goats fed an exclusive ration of lechuguilla before and after toxic effects developed.*

GOAT		DATES OF BLOOD STUDIES				
		2-28-33	3-7	3-11	3-14	3-17
62	R. B. C.	18		23		26
	Whites	11,600		11,200		17,400
	Polys	38				69
	N. P. N.	32	60	107	82	65
63	R. B. C.	20		19		23
	Whites	6,600		9,000		27,000
	Polys	23		72		72
	N. P. N.	33	88	107	120	139

R. B. C. = Erythrocytes, expressed in millions per c m of blood.
Whites = Leukocytes, in thousands.
Polys. = Polymorphonuclear leucocytes, percentage.
N.P.N. = Non-protein nitrogen.

gests that light conditions during this season of the year are more favorable for the development of resistance than for the development of an injury to the blood-vessels and a visible reaction.

Photosensitization in this disease may be of two sources; first, direct or that produced by the photodynamic principle in the plant and which is not dependent upon a liver injury and second, indirect or that produced by phylloerythrin, which may gain access to the peripheral blood as a result of the liver injury. A sensitization of this nature is in keeping with the results of Quin[3] and Rimington and Quin[4] who produced an obstructive jaundice followed by photosensitization, by ligating the common bile ducts of sheep and goats. The photosensitization in these animals was shown to be due to the presence of phylloerythrin in the blood stream, a porphyrin which is produced in the digestive tract of normal animals, but converted to a non-toxic substance by the normal liver. It should be mentioned that the photodynamic aspect of lechuguilla poisoning is of minor importance, as the mortalities are due to the toxic action of the hepato-nephro-toxin, especially in relation to the kidneys.

SUMMARY

An important disease of sheep and goats in the Southwest which is caused by grazing on the leaves of lechuguilla is described. The complete clinical syndrome of this disease presents the combined action of a photodynamic agent and a hepato-nephro-toxin.

The disease was reproduced by feeding the leaves of the plant to sheep and goats under experimental conditions.

Both aqueous and alcoholic extracts of the plant were found to be toxic. An uncomplicated photosensitization was produced in sheep by feeding the hydrolyzed products of the alcoholic extract.

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National Dog Week

As previously announced, the week of September 18-24 has been designated as National Dog Week by the leaders in this movement who maintain national headquarters at 3323 Michigan Boulevard, Chicago.

Small-animal practitioners should be particularly interested in this event, for plans have been made for extensive local publicity in many of the large cities throughout the country.

Government Trailer Laboratory

The first government built trailer to be used as a laboratory in the control of Bang's disease has been assigned to Michigan.

Clinical Laboratory Methods in Small Animal Practice*

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Several years ago, a clinical laboratory was established in the Raritan Hospital for Animals at New Brunswick, New Jersey, with the hope that it would be valuable in small-animal practice. This laboratory has developed into one of the most important departments in the hospital. It has become indispensable. A demand for information concerning laboratory findings in the small-animal clinic has grown with the increase in the number of laboratories of this type. For that reason, the data collected at the Raritan Hospital for Animals will be analyzed, and the results published in monograph form in the near future. This book will include data in blood chemistry, urine chemistry, and hematology. Other phases of the clinical laboratory may be included if time permits.

Until more information, based upon laboratory experience in the small-animal hospital, is made available, the veterinarian must go to the highly developed clinical laboratory in human medicine for assistance in interpretation of results. Data collected at the Raritan Hospital have convinced us, however, that many errors can be made in applying information obtained in human medicine to the dog. Some of the most outstanding of these errors will be discussed below.

LIVER DAMAGE IN THE DOG

The concentration of uric acid in the normal blood of man is said to vary from 1 to 4 mg per 100 cc of blood. In the blood of a normal dog, however, there is never much more than a trace of uric acid. Except in the Dalmatian coach hound, the highest concentration of uric acid which we have encountered in dogs which were apparently normal is 0.7 mg per cent. Benedict[1] has demonstrated that the end-product of purine metabolism in the Dalmatian is uric acid but Bollman and Mann and coworkers[2-4] have shown that in other dogs almost all of the uric acid is converted into allantoin by the liver.

Destruction of liver tissue results in an increase in the blood uric acid as well as in the amount excreted in the urine. For that reason, they believe that the amount of uric acid excreted in 24 hours is a good measure of liver function. Liver damage is indicated if more than 15 mg of uric acid per kilogram of body weight is excreted in that period. This is true even if the animal is given a diet fairly rich in purines, which are the precursors of uric acid. If liver damage is severe, there will be an accumulation of bile pigment in the blood and a typical jaundice will develop. Under these circumstances, the blood will give an "indirect reaction" in the van den Bergh test. In the dog, however, this test is seldom obtained unless the liver damage is so severe that recovery is unlikely.

In order to detect the onset of liver damage in the dog, therefore, uric acid determinations are most helpful. Ascites may develop in animals with liver damage, due to reduction in the concentration of serum albumin in the blood. Sometimes this edematous condition is diagnosed incorrectly as due to other abnormalities, such as kidney damage.

KIDNEY DAMAGE IN THE DOG

Kidney damage in the dog seldom follows the same course as it does in man. Severe cases of albuminuria in the dog are not common, so that blood proteins are not lost through excretion and edema does not often develop. In fact, animals with slight renal insufficiency are usually suffering from tubular damage. There is seldom any al-

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bumin in the urine and the kidneys of most of these dogs can concentrate the urine to a normal specific gravity. These cases can be identified best by a rise in the blood urea nitrogen and a lowering of the urea clearance. Thus, the common practice of diagnosing kidney damage in man, by the presence of albumin in the urine and by a lowered specific gravity of that excretion, would not detect these cases.

As kidney damage in the dog becomes more and more severe, albumin may or may not appear in the urine. The ability of the kidneys to concentrate the urine does decrease, however, so that the maximum specific gravity is below the normal range. The term "maximum specific gravity" is used to refer to the highest specific gravity which can be produced. A dog, therefore, with a low maximum specific gravity of the urine and a high blood urea, is generally suffering from severe kidney damage regardless of the concentration of albumin in the urine.

The apparent concentration of creatinine in the blood of the dog does not differ very much from that found in man. It is sometimes claimed that blood creatinine determinations and creatinine clearances in man are good measures of kidney function. We have found that the concentrations of creatinine in the blood of the dog do not increase much with kidney damage until it is severe. For that reason, creatinine determinations of the blood have little clinical value in the routine diagnosis of renal insufficiency. A marked increase in the creatinine concentration of the blood of the dog generally means, however, severe kidney damage.

Hyperglycemia in the Dog

When the analyses are made eight to ten hours after feeding, the concentration of glucose in the dog's blood will be similar to that found in man. Hyperglycemia, however, is not so common in the dog as it is in man. Glycosuria is also rather rare, since the renal threshold for glucose is not often exceeded. Diabetes mellitus is occasionally found, however, and it is not difficult to diagnose, especially if a sugar tolerance determination can be made.

Ketosis in the Dog

It is not surprising, therefore, to find little mention of ketosis or the accompanying acidosis in this animal. It may be that acetone bodies could be produced in starvation after the stored glycogen has been exhausted and the animal forced to subsist upon fat and proteins. We have not encountered such a situation. However, we have been unsuccessful in our attempts to produce ketosis in dogs when they were put on so-called ketogenic diets. Incidence, therefore, of deranged carbohydrate and fat metabolism in the dog is much less than in man. If acetone bodies are encountered, they probably are generally produced by a severe diabetes mellitus.

Specific Gravity of the Urine and Tests for Reducing Substances

Sometimes a laboratory will report a positive test for sugar in the urine of normal dogs. This is almost always due to the fact that the urine of dogs is more concentrated than it is in man. For that reason, reducing substances other than glucose, which are normally in urine, are so concentrated that they affect the copper reagent used to test for reducing sugars. The specific gravity of the urine of the dog may be as high as 1.060 and still be normal. This concentration effect can be overcome either by using less urine in the test, or better, by diluting the urine to a specific gravity of 1.020 or less.

Hematology in the Dog

Other differences between the laboratory findings in dogs and man could be cited. For example, the normal white blood-cell count in the dog is approximately 11,000, which is just about twice that found in man. We have observed increases in the count when infectious diseases were present in the dog. Few cases of leukopenia have been encountered, however, except with virus distemper. Leukopenias resulting from other causes are probably not so common in the dog as in man. Probably most cases of anemia in the dog are secondary in nature. No case of Addisonian an-

emia (formerly called primary pernicious anemia) has been diagnosed nor has a report of a case, occurring spontaneously, been found in the literature.

SUMMARY

The need for a text in clinical laboratory methods based upon experiences in a small-animal clinic is emphasized. Some of the differences between laboratory findings and interpretations in small-animal practice and those in human medicine are discussed. These differences should be a warning not to proceed too rapidly in any attempt to carry over the interpretations of clinical laboratory determinations from the human hospital to the small-animal clinic. They demonstrate the need for a study of the dog for the sake of the dog.

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The Gay Wag

When bosses grow censorious
And jobs are most laborious
We moan about the "dog's life" that we lead
And sigh and murmur ruthfully,
Although, to state it truthfully,
A "dog's life" is a pleasant life indeed!

A "dog's life"—with no worryings
At economic flurryings
Or any of the woes at which we flinch;
No debts to vex and harass him
No duties to embarrass him—
A "dog's life?" Why, a dog's life is a cinch!

He knows a smug felicity
Whose comfort and simplicity
Are complicated only by his fleas,
With never toil or sweltering
He gets his food and sheltering
And lives a life of sybaritic ease.

There's no responsibility
To trouble his tranquility
A dog's life is as joyous as a song;
I'd swap with him most cheerfully
But—'twould be cruel, fearfully,
To make *him* lead a Man's Life very long.

Berton Braley.

Many persons after once they become learned cease to be good; all other knowledge is hurtful to him who has not the science of honesty and good nature.—Montaigne.

The Treatment of Heart-Worm Infestation*

By C. E. BILD, *Hollywood, Fla.*

The treatment for heart worms in the dog is a relatively new subject in the field of veterinary medicine. Since the treatment of this condition is not without a percentage of complications and mortality, there are, and perhaps always will be, differences of methods used. There is very little literature available on the actual treatment and the handling of complications arising from filarial infestations.

LABORATORY DIAGNOSIS

Laboratory diagnosis of heart worms is a very simple procedure. In routine work, we have never used any method of diagnosis other than the quickly examined, fresh-smear method, no cover-slip being used. However, one may examine the serum of a blood sample after the blood has clotted. The larvae from the serum, at room temperature, are active up to 48 and even 72 hours.

A third method, advocated in recent literature, is the centrifuge method. This consists of adding one part of blood to two parts of a 1 or 2 per cent acetic acid solution and centrifuging. The dead larvae will be found in the sediment. Some men advocate adding a small amount of methylene blue to this acetic acid solution to stain the larvae partially. Such staining apparently does not improve observation of the parasites. Slides from either centrifuge samples mentioned seem to deteriorate, the larvae being barely visible at the end of 24 hours.

There is no virtue in night examinations of suspect blood. Since it is satisfactory, and very easy, to take blood for examination from any of the external leg veins, we have never attempted to check the possibility of any difference in blood from a superficial vein as against blood from skin capillaries. Most of our heart-worm cases are found in dogs over one and one-half to two years old, and especially dogs that live outdoors and are exposed to mosquitoes. We have seen exceptional cases of heart-worm larvae appearing in a dog a year old and once, on postmortem examination, found the mature parasite in a ten-month-old greyhound.

We also find it advisable to check the urine of prospective heart-worm patients. We check it for albumin, sugar, specific gravity and pH reaction. We make some exception to this rule in females, especially toy females, because they are too difficult to catheterize. Any atypical condition of the urine is most likely to be found in old dogs, especially when in poor condition.

Albumin is most commonly found in atypical urine. Except where albumin is found in small amounts, and readily clears up, these dogs do not satisfactorily respond to treatment. In these patients, it is very much worth while to attempt to overcome the albuminuria by putting the patient on a high carbohydrate diet, such as skimmed milk or buttermilk, cereals, vegetables and small amounts of dog biscuit. A preparation like Vita King and a little Karo syrup adds to the value of these diets and makes them more palatable. An albuminuria may often be decreased by supplementing the above diet with a daily or twice daily intravenous injection of glucose, buffered with insulin. If much of such treatment is necessary, I would question the advisability of recommending treatment for heart worms.

We find sugar in the urine so seldom that it has not been a problem. Specific gravity usually runs between 1.015 and 1.040. Occasionally, we find a case of polyuria with a specific gravity of 1.010 or less. Such dogs are not good subjects for treatment. Postmortem examination of these dogs usually shows degeneration of the kidneys.

The pH reaction of the urine has been of no great importance in our examinations. It is usually acid and normally so, but when alkaline in otherwise normal urine, adding sufficient meat to the diet usually changes the reaction back to acid. If desirable,

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small amounts of boric acid or ammonium chloride may be used to acidify the urine.

FACTORS INFLUENCING TREATMENT

As we all know, the treatment of heart worms must be a clinical problem. It therefore follows that one of the most important phases of the routine is the examination of the patient before the treatment begins.

It quite often happens that heart worms die spontaneously, causing the dog to have what we will call a heart-worm toxemia. This toxemia is due principally to absorption of the dead worms, which are foreign protein. Heart-worm toxemia may be found in any degree. It may be slight, as found during a light reaction at the beginning of a course of treatment, or it may be severe, caused by all of the parasites dying spontaneously. The degree of toxemia depends upon the number of parasites that the patient harbors.

I will discuss this toxemia later. There is a possibility that any dog presented for examination, showing filaria larvae, has a heart-worm toxemia. After all, most dogs are presented for examination because the owner has some reason to believe that it is not normal. With this in mind, we do not make a practice of starting the treatment for heart worms the same day we discover the larvae.

Age is an important factor in whether or not to treat a dog for heart worms, since we know that very old dogs do not readily respond to treatment. However, it quite often happens that a dog, seven or eight years old, responds to the treatment much better than a dog three years of age. The reason will usually be found in the examination of the patient before treatment. It follows that the better the physical condition of the patient, the better the chances for an uneventful recovery.

If we have reason to believe that the patient has no heart-worm toxemia, or other serious condition, we check the patient for intestinal parasites and remove them if we find any evidence that they are present. We then like to rest the patient about a week before starting the treatment, being sure that he has an ample diet with sufficient vitamin content. Patients in poor condition, especially those having had many hookworms, which accounts for some of the anemia usually found in heart-worm dogs, will do better to have their build-up period prolonged. We do not make a general practice of attempting to treat this anemia parenterally, though we frequently prescribe iron, quinine and strychnine in a liver-iron tonic for oral administration.

TREATMENT

The commonly used drugs in the treatment of heart worms are the antimony salts filsol and fuadin. It is to be expected that each manufacturer claims superiority for his product, but from my observations and correspondence with Florida veterinarians, it appears that each drug has about an equal number of followers. Of twelve Florida veterinarians I contacted, who have had considerable experience in handling this condition, four use fuadin and six use filsol exclusively, and two use filsol as the drug of choice, except in small toy breeds.

Both products are good. Fuadin is advocated, used either intravenously or intramuscularly. All but one of these experienced men I contacted use this drug intravenously, except as mentioned above, *i. e.*, when it is to be used on small toy dogs or very old, debilitated patients. Filsol must be used intravenously. I have treated about an equal number of dogs with each product and have never observed any difference in toxicity to the liver and kidneys. Large injections of either drug into puppies will show, on postmortem, a definite liver and kidney degeneration.

In many cases, the treatment of heart worms will show no apparent difference as to the superiority of either drug. However, filsol and fuadin differ in one major respect. Fuadin sometimes appears to free the peripheral circulation of larvae with less toxicity, the action of the drug apparently being only to sterilize the female filaria which may remain alive and persist in the heart, or at some later time go to the lungs or occlude the pulmonary veins. It is possible for these dogs, when given periodic examinations, to check free of the

larvae and apparently be normal in every way, for a period of from a few months to a year. Such dogs may suddenly become ill, show a very labored breathing and die. These non-larval-producing heart worms will be found in the pulmonary veins. Apparently the transition from the heart to the lungs occurred at the onset of the labored breathing.

I have never observed encapsulated heart worms macroscopically, except on one occasion. Filsol sometimes appears to cause the dog to show more reaction during the treatment, but the dogs which have been treated with filsol and check free of the larvae will be free of heart worms at any reasonable time later. In handling many dog cases, such as car accidents, poisons, and the destruction of old dogs, one has an opportunity to observe these postmortem findings. The added toxicity occasionally found from the use of filsol as mentioned above, is perhaps due to greater absorption of the parasites. This absorption is very desirable.

Antimony is reputed to be a drug much like arsenic except that it is not cumulative in action and is more readily eliminated, principally by the urine. Continued administration of antimony apparently does not lead to tolerance of the drug. Using filsol or fuadin, one notices that there is often a very marked difference in patient tolerance of the drug. For that reason, the dosage used by experienced practitioners varies with the patient.

Intravenous administration is our method of choice. Such a statement is open to some question, as previously mentioned. Some men maintain that in old dogs, and dogs that are apparently not good subjects for treatment, intramuscular injection causes less shock to the patient, thereby enabling the practitioner to extend the course of treatment over a longer period of time. Clinical trial, however, will prove that if it seems advisable to treat a dog for heart worms over an extended period of time, *i. e.*, three or even four weeks, and this practice need not be unusual, the drug can be given intravenously in small doses and with less fear of untoward results.

We treated an eleven-year-old German shepherd dog for heart worms this summer that was an exception to our usual routine for handling such cases. This dog was given daily injections of filsol intravenously. The period of treatment was four weeks. Three times during this period, the treatment was discontinued for a few days because of slight reactions. I think that the intravenous route is also to be preferred for small toy dogs. For us, a ½-inch, 23-gauge, long-bevel needle solves difficulties often encountered when continued intravenous injections are given to these small dogs. This needle is not flexible.

Opinion is about evenly divided among a dozen Florida veterinarians as to whether to treat filaria cases every day or every other day. The filsol pamphlet states that this drug should be given intravenously and not more often than every other day. In the fuadin pamphlet, this drug is recommended as best used intramuscularly, on a schedule of six injections a week. When used intravenously, they recommend that the preparation be injected every other day.

We have always treated filaria cases intravenously. For the past two years, we have treated all of our patients every day except, of course, those that show reactions. The treatment is temporarily discontinued in such cases.

Simply stated, the treatment of heart worm in the dog consists of giving a series of intravenous injections which cause death of the parasite and disappearance of the larvae from the circulation. When the parasite dies, it is usually absorbed, the absorption causing a toxemia. It is advisable that the patient be not called upon to handle this necessary absorption in too short a period of time. This last statement, like most of the statements made in this paper, must be made with reservations, because of the varying clinical problems that the treatment of heart worms presents.

Attempting to treat a dog in a short period of time this summer, I gave a large Airedale female, which showed a dozen larvae in each miscroscopic field, four injections totaling 15 cc of filsol in four days.

The dog showed pronounced reaction for three days, as evidenced by sluggishness and fever. She was given 20 cc daily injections of glucose during the period of reaction. The following day no larvae were found on smear examination. She has checked clear of larvae and has been in very good condition since that time.

The course of treatment in average cases where the patient is in good condition, usually lasts two weeks. However, in very heavily infested dogs and in old dogs, the course of treatment usually requires three weeks. In those rare cases where the dog does not tolerate the drug well, it is much better to extend the treatment over a month than to help the client get another dog.

Whether or not filaria cases are kept at the hospital has always been optional with the client. Except during severe reactions, such patients seem to do as well at home as in the hospital. We familiarize the client with the symptoms of a reaction from the treatment. He keeps us informed each day regarding the appetite and spirit of the patient. Such information indicates the probabilities of a reaction even before a temperature rise is noted.

Bearing in mind the idiosyncrasies of different patients to the drugs used, it is always best to start the treatment with small doses and increase the amount each day until the suspected maximum dose is reached, or the patient shows a reaction. It is very difficult to recommend a scale of dosage, because of differences in degrees of infestation, general condition of the patient, and tolerance to the drug. One can be safe in using the scale of 1 cc to each 25 pounds of weight for the initial dosage, but not over 2.5 cc as the maximum initial dose. The next three or four succeeding doses may be increased gradually to about 1 cc for each 20 pounds.

At this point, an additional increase of dosage is advisable, if used with care. The increased dosage may be stepped up to 1 cc for each 15 pounds, or, in some cases, 1 cc for each twelve pounds of weight with 5 cc as the maximum dose in very large dogs. Under this classification, small Pekinese and such toy breeds receive 0.25 cc, or less, for the initial injection. I started a six-pound Pekinese with 0.2 cc and increased the dose to 0.6 cc for the last of seven daily injections, following which, smear examination showed the patient free of heart worms, nor has he shown any evidence of infestation for eighteen months. No reaction attended the treatment given.

Boston, wire-haired and Scottish terriers, and other breeds of that size, would receive 0.5 to 0.75 cc as an initial injection and 1.5 to 2 cc as a maximum. Springer spaniels and large fox terriers would be started at 1 to 1.5 cc and the dosage increased to 2, 2.5 or 2.75 cc. For hounds, pointers and setters, we start the dosage from 1.5 to 2 cc and increase the dosage to 3 or 3.5 cc. Occasionally, we have given these dogs 4 to 4.5 cc. Five cc is apparently the maximum dose for the largest dogs. However, as stated above, when a patient is handicapped by advanced age, poor general condition, or evidence of albuminuria, we try to extend the course of treatment over a few weeks. This is best accomplished by the use of a lowered scale of dosage.

REACTIONS

If a reaction appears at any time during the course of a treatment, the treatment should be discontinued until the patient is apparently normal. However, some veterinarians, when treating light infestations, will give the dog one more injection after the appearance of a slight reaction, because they know that usually after a good reaction, though not necessarily a severe one, the patient will check clear of the larvae and will stay clear.

In treating heart-worm cases, we never weigh the dog in order to calculate dosage. We simply begin with a small initial injection and increase the dosage according to our estimate of the ability of the dog to stand the treatment. Then we continue the injections until a reaction appears or the dog checks negative.

It is good practice to make a blood smear before each injection. Quite often, after the first few injections, there will be an increase in the number of larvae before a decrease is noted. This is a good sign, and one that shows that the drug is working.

Daily examination of blood smears give one a good idea of the progress made by the patient.

About two-thirds of all of the dogs that we treat for heart worms show a reaction, either during the treatment, at the end of the treatment, or both. In very rare cases, a patient will show all the symptoms of prostration within an hour or two after the injection of the drug. This prostration is due to antimony poisoning and is best treated as such. We like to give sodium thiosulfate intravenously, followed by glucose. Camphorated oil is a good heart stimulant. A warm saline enema seems to be in order. It is important to keep the patient warm. We have noted this reaction only a few times, but not once in the last two years. With extreme care, such patients may be put back on treatment after a rest of at least two weeks after they are apparently normal.

The reaction usually found during the treatment of heart worms is, as we have mentioned before, due to absorption of the dead parasites. This reaction is manifested by any one, or all, of quite a number of symptoms, the first of which are usually a lack of spirit and a rise in temperature. Rise in temperature is not a constant symptom, nor is the degree of elevation. Frequently, vomition presages the onset of a reaction. Dogs under treatment, and especially those showing reactions, drink increased quantities of water. This fluid intake should always be encouraged.

Dogs usually show varying degrees of sluggishness during a reaction, and many of them show a stiffness of gait. This stiffness is apparently a general muscle soreness. It may be so severe that the patient can hardly rise and walk, but it only rarely develops into a paralysis. If the reaction is intense, the patient is very apt to go partially off feed or refuse food altogether. It is usually not a serious matter when a patient refuses food for a day or two, or even three days, provided that he is not allowed to dehydrate, or show prolonged dyspnea. It may happen, when a patient is very prone to reactions, that he will show an albuminuria during the reaction, even though the urine was albumin-free when the treatment was begun. In such cases, the patient should be given glucose, the meat content of the diet reduced, and the treatment discontinued until a few days after the urine is free of albumin. In the event that the urine does not check clear of albumin, I would advise discontinuing the treatment permanently. Another type of reaction found is the swelling of the head, particularly the muzzle, and swelling of the scrotum. As stated above, these symptoms of a reaction may be found with or without any of the above other symptoms. Personally, I think that the swelling of a dog's muzzle or scrotum is a very good sign, because it indicates that the antimony injections are finished. I can not remember of one such patient having succumbed to this treatment during the past four years.

The reaction symptom which I dread most is to find a dog running a few degrees of fever and showing an embarrassed respiration. He may or may not be off feed at first, but if this difficult breathing continues, the dog will show a clinical picture of pneumonia with a copious discharge from the nostrils. The fever usually persists. With good care, these dogs may be kept alive from one to three weeks, although growing progressively thinner and weaker. In such cases, the larvae are almost always absent from the circulation at the onset of dyspnea, and may have been absent for three weeks prior to the post-mortem which probably will be held.

Another rare reaction, and one that I have observed only twice, is evidenced by bile in the urine. Both patients were Boston terriers, about six years old. One of them displayed this symptom a month after the heart-worm treatment and died of jaundice. The other patient showed this symptom two weeks after the disappearance of the larvae, during which time he showed a slight albuminuria, diarrhea and sluggishness, but no fever. This dog was placed on a high carbohydrate diet and given glucose intravenously. He was also given 50 cc of blood twice intraperitoneally because of inappetence. The condition persisted a month.

In the event that treatment is temporarily discontinued because of reaction, it is well to check the blood daily. This routine gives some idea as to the course of the treatment. In patients that show very few larvae before the treatment begins and the larvae disappear from the circulation without the patient having a reaction, it is in order to give one or two injections after the disappearance is first noted. To be sure that such a dog is free of larvae, check the blood at daily intervals for a few days and at seven- to 14-day intervals for a few times.

For the treatment of heart-worm reactions, we follow a set routine. The antimony is discontinued, the patient is given mineral oil with a small dose of hydrochloric acid and a rennin-pepsin compound. It is a habit with us in all of our oil administrations to render the oily preparation tasteless so that the patient will swallow it. A warm saline enema is to be recommended which apparently helps to alleviate the stiffness. Daily oil and enemas are in order during the reaction. If this reaction is of any degree of severity, we like to administer calcium gluconate intravenously at the onset of the reaction. We also like to use calcium dextrose solution, with a small amount of phosphorus added. This preparation differs from other calcium compounds in that only rarely will it cause vomition. Should the reaction persist for more than twelve to 24 hours (and it usually does), we give dextrose intravenously and buffer the dextrose with insulin. The reaction may cause dehydration, especially if there is a diarrhea, or where the patient is too sick to want to drink water. This dehydration should always call for the use of saline and dextrose. Such saline and dextrose should be given intraperitoneally or subcutaneously. Using 2.5 per cent of glucose in normal saline, it may be given in daily amounts of 5, 10 or 15 cc per pound of body weight, or as indicated. It is sometimes necessary, in severe heart-worm toxemias, to administer glucose in addition to the glucose in the saline. Regarding the total amount of glucose to be given daily, 1 or 1.5 cc per pound of body weight of a 50 per cent solution is in order. I am aware that in human medicine it is not uncommon to double this scale of glucose dosage. Such glucose is best given at intervals during each 24 hours.

SUMMARY

1. The direct smear method of heart-worm diagnosis is satisfactory and accurate.

2. Check the patient as thoroughly as possible before beginning the treatment.

3. It has been found best to treat dogs intravenously every day.

4. Start the treatment with small injections and increase the dose gradually.

5. Check the temperature and blood before each injection and extend the course of treatment if desirable.

6. Many reactions during treatment for heart worms may be anticipated. It is therefore advisable to be prepared.

7. The mortality rate may be kept down to 2 or 3 per cent.

Domestic Animal Diseases Produced by Light*

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The following is an account of the diseases of domestic animals which have been most clearly demonstrated to be produced by sunlight. It will attempt to show how certain laboratory findings bear directly upon problems which these diseases present, and to point out to what extent their etiology is established.

Since light is the precipitating factor in these diseases, the general properties of light must be kept constantly in mind in their study. This requires a somewhat different viewpoint than that from which diseases produced by microörganisms or by toxic substances are regarded. I wish, therefore, to mention certain elementary facts about light and its biological effects to which I will have to refer in the course of the following discussion.

In figure 1 is shown a map of that part of the spectrum which will be of interest to us here. This includes a region of wave-lengths extending from the short wave-length limit of sunlight, which is slightly below 3,000 Å, through the region of human vision, which extends from about 3,900 Å to about 7,000 Å, and slightly beyond. We refer to those wave-lengths which are perceived by the human eye as visible; to wave-lengths shorter than this as ultra-violet; and to longer wave-lengths as infra-red. These regions, which have no sharp limits and no real significance, should be attached to these divisions which are made only for convenience.

In the figure, a curve is drawn to represent the spectrum of sunlight, *i.e.*, the "average" distribution of the energy of sunlight with regard to wave-length. There is also a curve which shows the relative effectiveness of those wave-lengths which produce sunburn in human skin. Only wave-lengths shorter than about 3,300 Å cause sunburn, and it will be noted that only a tiny corner of the spectrum of sunlight is capable of producing this effect. This small corner is an extremely variable part, being effectively filtered out by smoke and dust, and this accounts for the great variability in the length of exposure leading to sunburn.

A third curve in figure 1 represents the spectral transmission of window glass, showing that it cuts out virtually all the wave-lengths which are responsible for sunburn. This shows why one does not sunburn through window glass. The same region which causes sunburn, *i.e.*, wave-lengths shorter than 3,300 Å, is destructive to living organisms in general, and is capable of producing symptoms similar to sunburn in mammals other than man. Although I am not sure of this, I believe that such "sunburn" may be often troublesome in domestic animals, and may represent some of the symptoms attributed to photosensitization from feeding on plants.

I have called attention to this region of sunburn and other destructive effects mainly by way of contrast to the diseases that I wish to discuss, produced by light outside this general region, and which have as little relationship as possible to ordinary sunburn. It is necessary to have this point clearly in mind, because a certain confusion of ideas has arisen in this regard.

Sometime early in the last century, the idea was current that only a part of the spectrum, comprising roughly the ultra-violet, violet and blue, was capable of producing photochemical reaction. This may have come from the fact that the unsensitized photographic plate is sensitive only to these wave-lengths. The terms "chemical rays" and "actinic rays" came from this concept. More recently, the term "ultra-violet rays" has been used in much the same sense. Such ideas have absolutely no experimental or theoretical basis.

*Presented in a symposium at the California Veterinary Conference, Davis, Calif., January 5, 1938.

It might be pointed out that more photochemical reactions are produced by shorter than by longer wave-lengths, and that there is a long wave-length limit in the infrared beyond which no photochemical reactions are to be expected; but I do not wish to take time to discuss the reasons for this. I must, however, point out that the terms "actinic rays" and "chemical rays" have no meaning, and that use of the term "ultraviolet," to designate a supposed region in which all photobiological effects occur, is absolutely false. The sooner these concepts are forgotten, the faster will progress be made in the explanation of photobiological effects.

There seems little doubt that at least the more important diseases in domestic animals produced by light are examples of photodynamic action, a phenomenon which has been frequently studied in the laboratory. The following is an example: If red blood-cells are freed of serum, suspended in saline solution and exposed to sunlight in a glass tube which cuts out destructive short wave-lengths, the cells remain undamaged for several hours at least. If a small quantity of any one of a number of dyes, *e.g.*, eosin, is added to the suspension, the cells may be completely hemolyzed in the course of a few minutes. Similar destructive effects have been produced in a wide variety of living organisms by the use of a considerable number of dyes.* Of greatest interest to us here is the fact that laboratory mammals, *e.g.*, mice and guinea pigs, when injected with such dyes and exposed to light, show symptoms which are comparable to those observed in domestic animals suffering from Saint Johnswort or buckwheat poisoning.

Such effects have been confused with sunburn and those other effects which are produced by ultraviolet radiation shorter than 3,300 Å. Actually, they have little or no relationship, the clearest point of distinction being that the photodynamic effects occur only in the presence of molecular oxygen, while the other effects take place in its absence. Experimental evidence for this is presented by Blum and Spealman[1] and by Blum, Watrous and West.[2] The wave-lengths which produce the photodynamic effects vary according to the dye used, in accordance with a fundamental law of photochemistry known as the Grotthus-Draper law, which states that only light which is absorbed by a system may pro-

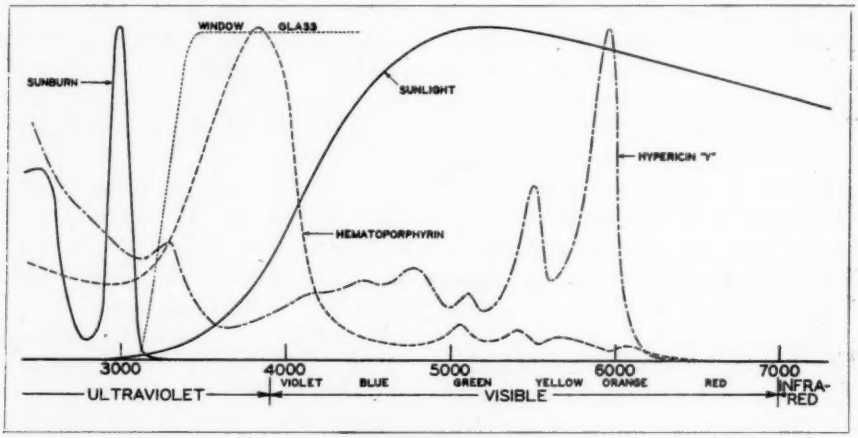

FIG. 1. Map of part of spectrum.

Abscissa=wave-lengths in Å.
Sunburn=relative effectiveness of various wave-lengths for producing the erythema of sunburn.
Window glass=transmission of a sample of ordinary window glass.
Ordinates=arbitary values chosen so that maximum values have the same ordinate.
Sunlight=distribution of the radiation in "average" sunlight.
Hematoporphyrin=absorption spectrum of hematoporphyrin hydrochloride in aqueous solution.
Hypericin "Y"=absorption of one of the components isolated from Hypericum by Mackinney and Pace.

*For a further discussion of photodynamic action, see Blum (1932).

duce photochemical change in that system.

In most cases, there is only one substance in such a system which is the photochemically-active light absorber, and hence only the wave-lengths absorbed by that substance produce the photochemical changes. Every substance absorbs only certain wave-lengths of light, as shown by the different colors which are displayed. In the case of photodynamic action, it is well established that the dye is the substance which absorbs the light and produces the photochemical changes. Experimental evidence for this is discussed by Blum,[3] Blum and Scott[4] and Blum and Pace.[5]

Figure 1 shows the relative amounts of energy absorbed at different wave-lengths by two pigments which have photodynamic properties. Such curves are known as absorption spectra. If the absorption spectrum is known for a photodynamic dye, it should be possible to make a prediction as to what wave-lengths will produce the photodynamic effects when that dye is used; and, conversely, if the action spectrum is known, *i.e.*, the relative effectiveness of the wave-lengths which produce the photodynamic action, it may be possible to make a guess as to the photodynamic substance which is acting as the light absorber. There are numerous factors which prevent an exact agreement between absorption spectrum and action spectrum, which are discussed by Blum and Pace, but an approximate agreement is to be expected. As I hope to point out, this should furnish an important tool in establishing the etiology of the diseases produced by light.

POSTULATES FOR DEMONSTRATING A PHOTODYNAMIC DISEASE.

As I have said above, it seems certain that at least the more important domestic animal diseases produced by light are examples of photodynamic action, although it cannot be said that the etiology is absolutely established in any one case. I should like to formulate three postulates which I believe should be satisfied before such a disease can be regarded as proven to result from photodynamic action. It may be found, as in the case of Koch's postulates for establishing the etiology of a microbial disease, that it is not always possible to demonstrate all the postulates, but this should always be attempted.

Postulate I: The disease must be reproduced by exposure of the animal to sunlight, and it should be shown that the symptoms disappear when the animal is protected from sunlight. The symptoms will be discussed in more detail below. It will suffice for the moment to say that they include; (1) signs of sensory stimulation in the skin during the time of exposure to light, such as scratching, rubbing and running about, and (2) lesions of the skin which appear subsequent to exposure and which may persist for a long time. It is specified that sunlight must be used in demonstrating this postulate, because artificial sources may be deficient in the wave-lengths which produce the effects, and their use lead to apparently negative results. Sunlight is the radiation to which animals are exposed in the field and it contains all the wave-lengths which are of importance in such studies. This postulate should always be established before a disease is definitely stated to be produced by light.

Postulate II: A substance must be isolated which can be shown to produce the symptoms of photosensitivity when injected into animals. It is to be presumed that this substance will be, as a rule, of plant origin, and, for reasons to be discussed below, it must be a substance other than chlorophyll.

Postulate III: It must be shown that the wave-lengths, which produce the symptoms of photosensitivity after the injection of the substance isolated in compliance with postulate II, are the same as those which produce the symptoms of the disease.

The value of these postulates will become more clear in the course of the following discussion, where I shall show to what extent they are fulfilled by the experimental evidence in the various diseases of this type which are found among domestic animals.

HYPERICISM OR SAINT JOHNSWORT POISONING

This disease results from feeding upon certain species of *Hypericum*, most com-

monly *Hypericum perforatum*, which is called Saint Johnswort or, in California, Klamath weed or Tipton weed. It is called Hartheu or Johanniskraut in Germany, and Millepertuis in France. The first postulate has been demonstrated by many field observations, and by the controlled experiments of Dodd,[6, 7] Henry,[8] Seddon and Belschner,[9] and Quin.[10] The experiments of Marsh and Clawson,[11] which led them to minimize the importance of the disease in this country, do not seem very conclusive and are not upheld by the field observations of Sampson and Parker.[12]

The second postulate also has been satisfied. A substance, hypericin, which produces photosensitivity when injected into animals, may be isolated from the plant (Ray,[13] Melas-Johannides,[14] and Horsley[15]). The exact chemical nature of the substance is not established, and recent studies by Dr. Mackinney, of the College of Agriculture, University of California, and Mr. Pace, of my laboratory, indicate that the preparations used by earlier workers contained more than one substance. Their experiments, which are still in progress, indicate that there are at least two very similar photodynamically active compounds in *Hypericum* which are responsible for the symptoms of the disease.

The third postulate has not been established. It is being studied by Mr. Pace. The absorption spectrum of one of the components isolated by Mackinney and Pace is shown in figure 1, and this may indicate the wave-length region to which the animals suffering from hypericism are sensitive. However, the absorption spectrum varies greatly with hydrogen-ion concentration, in this instance, and may be very different in the skin than when in solution in organic solvents.

Granting that the third postulate will be shown to be satisfied, the etiology of the disease may be described as follows: The plant *Hypericum* contains a photodynamic substance which is not destroyed in the digestive tract, and passes across the intestinal wall into the blood-stream. It passes from the blood-stream to the skin, which it sensitizes to light in the same manner as other photodynamic sensitizers. The establishment of the second postulate does not prove this, because the mere finding of a photodynamic substance does not prove that it may pass across the intestinal wall and be carried to the skin by way of the blood-stream.

The demonstration that the skin is sensitive to the same wave-lengths if the photodynamic substance is taken into the digestive tract, or injected directly into the blood-stream, serves to establish this point. It must be pointed out that chlorophyll may be extracted from any green plant and will sensitize the animal to light if it is injected, but it is broken down in the intestinal tract and does not pass across the intestinal wall into the blood-stream. This is a very fortunate thing, for if it were not true, you and I would become sensitive to sunlight after every lettuce salad we eat.

GEELDIKKOP

This is the Dutch name given to what is considered the most important small-stock disease of the Karoo veldt in South Africa. The translation is "yellow thick head," which describes the most obvious symptoms of the disease, namely, swelling of the skin of the head and intense icterus.

The disease results from feeding on plants of at least three not closely related plant genera, *Tribulus* (Zygophyllaceae), *Lippia* (Verbenaceae), and *Panniсum* (Graminae) (Rimington and Quin[16]). The plant most commonly associated with the disease is *Tribulus terristris*, which seems to produce the disease only under certain climatic conditions. Ordinarily, it is one of the better forage plants of the Karoo, but when summer rains, which cause it to grow rapidly, are followed by drouth, it wilts, and it is at this time that it becomes toxic (see Quin and Rimington[17, 18]).

Several steps in the etiology have been quite clearly established by the beautiful experiments of Quin and Rimington,* but a few points remain to be demonstrated. These investigators have repeatedly shown

*The more important of the papers by these investigators are: Quin (1929,[19] 1933a,[20] b,[10] c,[21] 1936[22]), Quin and Rimington (1935[17]), Rimington and Quin (1934,[23] 1935[24]), Quin, Rimington and Roets (1935[18]).

that the first postulate is fulfilled. The second is also established but, in this case, the picture is somewhat more complicated than that of *hypericism*. The sensitizing substance appears to be phylloerythrin, which is normally produced in the intestine of animals feeding on chlorophyll-containing plants, by the breakdown of the latter substance. It is readily taken through the intestinal epithelium into the blood-stream, is collected in the bile and excreted back into the intestinal tract.

In this way, the concentration of phylloerythrin in the blood and hence in the skin, never reaches a high level, but if the excretion of the bile is prevented in any way, this substance gets into the skin and renders the animal sensitive to light. This was nicely shown by Quin, by tying off the bile duct of sheep and goats. If such animals were fed on chlorophyll-containing plants, they became sensitive to light, but if kept on a chlorophyll-free diet, they showed no such symptoms. Rimington and Quin found a toxic principle in *Lippia* which produced damage to the liver, so that icterus and photosensitivity resulted, the latter symptoms occurring only when the animals were fed a chlorophyll-containing diet. They have not yet isolated such a toxic principle from *Tribulus*, but it is presumably present in that plant.

The third postulate has not yet been fulfilled. Phylloerythrin is a porphyrin. These compounds all have similar absorption spectra. That for phylloerythrin not being available, the absorption spectrum of another porphyrin is shown in figure 1 to indicate the spectral region in which photosensitivity is to be expected.

FAGOPYRISM OR BUCKWHEAT POISONING

Although this is the best known of the diseases produced by light, its etiology is not so clear as those of the two diseases just discussed. There is little doubt about the first postulate, thanks to the experiments of Wedding,[25] Öhmke,[26] Merian,[27] Sheard, Caylor and Schlotthauer,[28] and Lutz,[29] although Bicklmaier[30] claims to have been unable to produce photosensitivity by feeding this plant. The experiments of Sheard *et al* show that some animals are much more susceptible than others.

As to the second postulate, the substance isolated by Öhmke, and by Fessler,[31] was undoubtedly chlorophyll (see Lutz[37]), and the fact that this produced photosensitivity, when injected, has little bearing on the etiology, for reasons discussed above. The "fluorophyll" isolated by Busck[33] in 1905, was probably a different substance, but unfortunately it has never received further study.

The third postulate must remain unsatisfied until the second has been demonstrated. However, there has been some suggestive study along this line. Sheard *et al.* found certain absorption bands in the serum of sheep fed on buckwheat which were not present in the serum of normal sheep. These occurred at 5,800 and 6,000 Å, and certain attempts to find the region of spectral sensitivity of animals fed on buckwheat seemed to indicate the same region. The latter measurements were not at all quantitative. Sheard *et al.* suggest that the photosensitizing substance may be phylloporphyrin or phylloerythrin, but their measurements would not agree at all with the absorption spectra of porphyrins. The etiology of *fagopyrism* can not be regarded as clearly established.

CLOVER DISEASE, ETC.

There are numerous reports of photosensitivity following the feeding of various leguminous plants, including lucerne, burr clover, Swedish clover and vetch. Dodd[34] performed a few experiments on guinea pigs which indicate that burr clover sensitizes these animals to light, but there seem to be no other experimental studies. It seems improbable that such important forage plants should produce photosensitization without its being more commonly observed, but there are things which suggest that this is a condition like geeldikkop, which occurs only when the plant is in a particular condition. On the other hand, it is doubtful if the icterus, which is a characteristic symptom of geeldikkop, is present in clover disease.

It has been suggested that other diseases have similar etiology, *e.g.*, maidism, which is supposed to arise in animals fed exclusively on maize, and a rash which is supposed to follow feeding on potatoes. There is some experimental evidence for the former, but I can find none for the latter. Certain conditions, which have been suspected as due to light, will be discussed by other members of the symposium, so I shall not mention them at this time.* I will try, however, to point out a few more laboratory findings which have bearing on these diseases.

Symptoms: Hausmann[35, 36] studied the effects of intensity of light and amount of dye (hematoporphyrin) injected, on the symptoms of photosensitization in mice. He describes the following degrees of effect: *A. Lightstroke.* When the light is very intense and the amount of dye very great, the animal may pass into a coma in a few minutes after a very short period of excitement and die without further symptoms. *B. Acute form.* Under less severe conditions, the animal scratches vigorously at first and waltzes about. In a few minutes the ears become inflamed and the animal blinks the eyelids and attempts to avoid light. Shortly afterward, he becomes weak and dyspneic. In some cases agonal tetany is observed and death may occur in a few hours. *C. Subacute form.* Severe edema of the skin occurs, but the other symptoms are less in evidence. *D. Chronic form.* With weak intensity of light or low dye concentration but repeated exposure, the ears may eventually become necrotic and slough off, but the other symptoms are absent.

With such a range in degree possible, it is not surprising to find the symptoms of these diseases somewhat differently described in the literature. Descriptions comparable to at least the three last forms described by Hausmann may be found. There is a possibility of missing the less severe degrees, yet they may be very important. For example, Pichon and Baissas[37] have described mild symptoms in catttle fed on buckwheat where no skin lesions were observable except those resulting from scratching. Irritation of the udder and teats was so great, however, as to result in occlusion of the milk-duct. In the case of *hypericism,* the losses from severe illness or death may be less important in the long run than losses of wool from minor skin lesions. Other symptoms may be secondary to the effects produced by light. Blindness sometimes occurs and this may result in death due to inability to find food. Inability to feed may also result from damage to the skin of the face and lips.

It is often thought that skin damage in these diseases is limited exclusively to the white areas. Hausmann[35] found that he could produce symptoms in gray mice of all but the most severe degree, showing that while the pigment of the skin may serve as an effective filter, it does not necessarily cut out all the light which causes the damage. Hence, the white areas of skin may be expected to be the most severely involved, but the colored areas should also be sensitive, though to a less degree. As a rule, the lesions are observed only on white areas, but this is not universally true.

IMPORTANCE OF THE DISEASES AND NEED FOR MORE FUNDAMENTAL WORK

I am sure that the papers which follow will serve to point out the importance of these diseases by indicating that their incidence is much more common than is ordinarily suspected. Certainly the problem of Saint Johnswort is an important one in California, as it is also in Australia. *Geeldikkop* itself may not be so far distant as the Karoo veldt. The plant which is principally responsible for it, *Tribulus terristris,* is none other than the "puncture weed" which is spreading rapidly in the western part of this country where, in some places, the climatic conditions must resemble those of the Karoo. Can we expect to find this disease occurring among sheep in these western states?

I hope that the above discussion has served to convince you that the studies which have been made in the laboratory have a direct bearing on the field problems

*These included photosensitization by Sudan grass (Howarth, 1931), big head in sheep (Clawson and Huffman, 1937), and photosensitization by *Agave lechugilla* (Mathews, 1937).

which these diseases present. I hope that you are also convinced of the necessity for further fundamental investigation in the laboratory. I may cite, as an example of what might be done, the finding of a small laboratory test animal which would be appropriate for the study of these diseases.

Suppose, for example, that a certain plant is suspected of producing photosensitization in the field, but that the collection and feeding of this plant to large stock animals is not feasible. If we knew that the chances of producing photosensitivity in a small laboratory animal were good, we might save much time and effort. If the chances were not good, we should not only be wasting time, but would be running the risk of drawing false conclusions. At the moment, we know very little about the relative sensitivity of laboratory animals to the various types of photosensitizing plants. The problem is therefore not nearly so simple as it might seem on superficial consideration. Here is at least one place in which laboratory studies could help. I am sure there are many others.

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Hog Income Report

Hog producers last year realized a cash income of $906,525,000 from sales of hogs, pork and lard, according to the third of a series of commodity income estimates by the Bureau of Agricultural Economics.

The Occurrence of Salmonella Newport in Domestic Animals*

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Salmonella newport was first recognized as a distinct entity by Schütze.[1] Since that time, it has been found more or less regularly by English and European workers who classified the *Salmonella* strains isolated from cases of food poisoning in humans. Its prevalence in Europe may be judged by the results of Kristensen and Bojlen,[2] who found 58 cultures of *S. newport* among 737 *Salmonella* strains studied. In their series it was exceeded in number only by *S. typhi murium* and *S. thompson* var. *berlin.*

Although it occurs frequently in food poisoning and is regarded as a species widely distributed in nature, little is known of the distribution of *S. newport* among the domestic animals. It is known to occur in swine, and Hormaeche and Salsamendi[3] found that, in point of number, it ranked fourth among *Salmonella* strains isolated from hogs in Uruguay.

As far as the writer is aware, the presence of *S. newport* in the United States has not been reported. Graham[4] reported the isolation of an organism related to *S. newport* from quail. Through the courtesy of Dr. Graham, a transplant of this organism was obtained by the writer and identified as *S. oranienburg.*[5] The purpose of the present paper is to report the positive identification of *S. newport* among strains isolated from domestic animals in the United States. In the identification of the organisms, the somatic antigens and the specific and non-specific phases of the organisms were identified by agglutinin absorption.

Four cultures of animal origin have been identified as *S. newport.* The sources of these cultures were as follows:

Culture 623: An unidentified bacillus in the collection of Dr. E. O. Jordan. Received by Dr. Jordan, in 1928, from Dr. L. M. Roderick, who isolated the organism from the spleen of a hog affected with necrotic enteritis.

Culture 21404: Isolated from the intestinal contents of a steer. This animal was one of a group of 50 heavy steers, several of which were affected with bloody diarrhea. Two animals died of the disease and the organs of the second were brought to the laboratory for examination. No coccidia were found in the intestinal contents. Bacteriological examination of the spleen and liver was negative. Large numbers of *S. newport* were found in the intestine, which exhibited hemorrhagic enteritis. The animals were shipped for slaughter before additional fecal specimens could be obtained.

Culture 21834: Isolated from the intestine of a hen affected with tapeworms and enteritis. This hen was one of three birds from a flock which had a high mortality rate for several months. All three birds were infested with tapeworms and had a chronic enteritis. Only one was examined bacteriologically. No organisms were isolated from the liver or spleen. Numerous colonies of *S. newport* appeared on brilliant-green plates inoculated with intestinal contents.

Culture 21913: Isolated from the intestine of a pullet from a flock which had suffered heavy losses. The birds were said to die suddenly, without exhibiting previous symptoms of illness. The pullet had a severe enteritis and was affected with coccidiosis. Tetrathionate broth was inoculated with material from the intestine. Numerous colonies of paratyphoid bacilli developed on brilliant-green plates inoculated from the enrichment medium. Examination of these colonies revealed the presence of two species which were identified as *S. newport* and *S. seftenberg.*

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†The investigation reported in this paper is in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director.

SUMMARY

The presence of *Salmonella newport* in infections of domestic animals in the United States is reported. The organism was recognized in hogs, cattle and chickens. In one instance both *S. newport* and *S. senftenberg* were recovered from the same chicken.

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What a Veterinarian Can Do to Improve Live Stock in His Community*

By R. W. WILLIAMS, *Eldorado, Ark.*

The veterinarian has a greater opportunity than any other citizen in his community to aid in the improvement of live stock. In fact, he should be the chief executive under whom all projects along this line are directed. The steps that he may take are too numerous to mention in a few words, but some of the most important ones are given below.

First in importance is the proper breeding of live stock. Unfortunately, this subject has been too long neglected. Explanations to farmers and ranchers relative to the true value of purebreds are always welcomed by live stock owners. Once converted, they become great enthusiasts, taking pride in purebred ownership and satisfaction in greatly increased financial returns.

Proper disease control comes next as an aid to the betterment of live stock, managed largely through prevention and treatment, such as periodic testing for tuberculosis and Bang's disease, and inoculations at regular intervals for the prevention of blackleg, anthrax and hog cholera. We must also guard against the various diseases that are transmissible from animals to man. Some of these diseases and sources are: From meat; such common diseases as tularemia, trichinosis, tuberculosis, botulism, tapeworms and various other meat-borne diseases, too numerous to mention. From milk; tuberculosis, undulant fever, septic sore throat, typhoid, dysentery and many others.

We, as veterinarians, should be pioneers in educating live stock owners regarding methods of prevention and eventual eradication of the above mentioned diseases. This we can do by advocating periodical testing and prophylactic treatment, teaching proper sanitation and rendering better veterinary service in a strictly professional, ethical way. Above all, it is advisable to discourage the use of nostrums, patent medicines and quackery of all kinds. On the other hand, we should be ready at all times to give such information as will inspire confidence, thus inducing live stock owners to ask advice of their best friends, the veterinarians.

Veterinarians have rendered a greater service in the control of live stock disease than can be estimated in terms of dollars and cents, for it was due to the initiative of veterinarians that suitable and necessary control laws were passed and enforced, resulting in the eradication or control of many communicable animal diseases, such as foot-and-mouth disease, for example.

It is the duty of every practitioner to encourage farmers to attend clinics once a year for the purpose of having their live stock examined and treated by competent veterinarians. By so doing, their live stock stay in better condition for work and have more resistance to many diseases.

Another way in which veterinarians may be of great service in the improvement of live stock is to educate owners concerning proper rations for animals. This object can be accomplished by the distribution of appropriate literature and by word of mouth. Calcium and phosphorous deficiencies are cases in point. Live stock owners can easily learn and practice methods for overcoming these deficiencies by correct feeding methods.

Lastly, let us not overlook the fact that our live stock is entitled to our protection from treatment by non-professionals. Such treatment puts a low value on veterinary medicine as a whole, and discourages the growth of the live stock industry.

Then there is the human side of the question. Ethical veterinarians are using anesthetics in increasing amounts. In cities, many dogs and cats are getting better med-

*Presented at the annual meeting of the Arkansas Veterinary Medical Association held at Hot Springs, March 7, 1938.

ical treatment than some members of rural families. Must rural practitioners sit idly by and keep silent while the brutalities of quackery replace modern, humane methods?

Farmers are fond of their animals and, as a rule, are kind to them. If they consent to unscientific methods of treatment, it is because unscientific methods are thrust upon them. An investigation should be made into what is transpiring along this line in rural communities and steps taken for correction of the evil.

Veterinarians can accomplish much toward improvement of live stock, but only if held in high esteem by the general public. Therefore, in order to improve the live stock industry, we must continually strive for the good-will of our fellow-man, which can be gained by professional and courteous manners. Then, by the exercise of our best professional knowledge, we may do our utmost to improve the live stock industry, which is so essential to the welfare of the nation.

Development of Veterinary Work in India

The earliest available official reference to veterinary work in India appears to be a general order of the Bengal army, dated April 8, 1793, "to order sick animals to be inspected by persons who may be judged capable of distinguishing the disorders of horses." At that time, there was not a single qualified veterinarian in India, and only two in England. In 1799, graduate veterinarians proceeded to India, but two were soon invalided home and little is known of their work. It appears that these veterinary officers were attached to British regiments serving in India. In 1823, J. T. Hodpa submitted to the Government of India a plan for the creation of an organized veterinary service.

In 1827, twelve veterinarians came out to India, and in 1828 a further 13, and subsequently, veterinarians were supplied as casualties occurred. The total number at this time was 31.

The next important event in the advance of veterinary work in India was the formation of an Indian Civil Veterinary Department, with schools for the training of students, in 1850. The first veterinary school opened in India appears to have been in 1874. Subsequently, veterinary colleges were established at Bombay, Calcutta, Madras and at Lahore.

The value of veterinary work gradually became established, but even today the position of disease control in India is peculiar, in that there are no veterinary practitioners, except a few in Calcutta and other big cities, and live stock owners are dependent almost entirely on government veterinary service for veterinary aid. It is therefore incumbent upon governments to organize and maintain suitable provincial veterinary services for the control of contagious diseases.—*The Veterinary Record.*

Will You Cooperate with the Acting Secretary-Editor in Securing New Member?

SWINE ERYSIPELAS IN NEWBORN PIGS*

By E. F. WALLER, *Ames, Iowa*
Department of Veterinary Pathology, Iowa State College

On June 7, 1938, there were submitted to this laboratory for examination three dead baby pigs with the following history:

History: Three days previously, on June 4, a large Chester White sow was noticed sick. She would not leave her nest, but since she was due to farrow, no particular attention was given her. During the night of June 6, 16 pigs were farrowed, four of which were dead. By 2 p. m., June 7, the three baby pigs, referred to above, had died. Upon examination, the following lesions were observed:

Postmortem: Erythema of the abdomen, snout, ears and tail; subcutaneous edema of abdominal wall; subperitoneal edema about the kidneys in one carcass, and enlarged pulpy spleens in two. Cultures made from the spleens and kidneys produced pure growths of *Erysipelothrix rhusiopathiae* after 24 hours of incubation.

On the morning of June 8, four more pigs from this litter had succumbed. The lesions observed were similar to those described in the first three pigs, varying only in extent. *E. rhusiopathiae* was isolated from two of this group. The farm was visited that evening and this sow was found to have lesions resembling those found in the skin form of swine erysipelas. She carried a temperature of 103.2° F. at this time.

Treatment: Swine erysipelas immune serum was administered to her by Dr. W. R. Anderson, of the Ambulatory Clinic staff. This was repeated in approximately 48 hours. At the time of this writing (June 21, 1938), she is apparently normal except for some skin lesions. Examination of the remainder of the herd revealed four additional breeding animals with either skin or joint lesions resembling those found in swine affected with a chronic form of swine erysipelas. One of these animals, a large Hampshire sow, has since died and, upon autopsy, revealed a vegetative endocarditis as well as the aforementioned lesions of the dermis.

Comment: Swine erysipelas did not become a major problem in Iowa until the spring of 1937. In 1936, only 14 cases were submitted to this laboratory, while in 1937 there were 111 cases. Prior to this time, the disease was limited to individual farms or communities. The greater part of these cases for 1937 were of the septicemic form and in pigs under three months of age. Bongert[1] mentions that the transmission of swine erysipelas from the sick sow to the fetus has been described. Van Es and McGrath,[2] in 1936, made the following statement:

> The susceptibility to swine erysipelas is least marked in the age group under three months, the preponderating majority of the cases falling within the age group of three to twelve months. It remains, however, possible that swine much older even at the age of three years, may contract the disease. Pigs of tender age have been observed to become infected although such cases are relatively rare.

The heavy incidence of the septicemic form of swine erysipelas in suckling pigs submitted to this laboratory in 1937 and so far this year does not bear this out. Possibly the large amount of the chronic form of this disease observed in breeding herds during the past fall and winter, as manifested by arthritis and skin sloughs, has played a large part in producing the heavy infection in young pigs.

*Received for publication, June 25, 1938.

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TETANUS IN A COW*

By CHAS. HAASJES, *Shelby, Mich.*

On September 14, 1935, according to my records, I was called to see a five-year-old Jersey cow that had freshened four weeks previously and had been off feed and stiff for several days.

History: The owner stated that the patient had a retained afterbirth following her last parturition, but would not call a veterinarian for such a simple job as that. He admitted that force had been used, which probably produced many lesions in the uterus. Further questioning brought out the fact that the animal had deficated several times while he was removing the retained placenta. As the fecal material was not promptly removed from the hand and arm, some of it probably was carried to the uterus.

Symptoms: When examined by me, the cow was standing in a stiff position with the back arched and the base of the tail raised. The least excitement caused the membrane nictatans to move back and forth over the eyeball. This occurred when an attempt was made to open the mouth, which was impossible to do, even with a wooden stick. The throat was paralyzed. Thick, white pus was discharging from the vagina. The uterus contained about a quart of putrid pus.

Diagnosis: Tetanus.

Treatment: As the owner insisted that it was not the rear end of the cow that needed treatment, I declined to proceed further with the case. Because I refused to prescribe drenches, the owner and his wife gave large doses of bromides in water per os. The cow died from mechanical pneumonia four days after I was called to see her. The neighbors saw the autopsy and reported to me that the lungs were in a bad condition.

Discussion: I believe that the feces of herbivorous animals commonly contains tetanus spores. In that case, there would appear to be little doubt as to how this animal became infected, especially as I could not locate a visible wound. However, is it not possible for infection to enter a needle puncture after the needle is withdrawn? If so, would not tetanus infection be possible following the intradermal T.B. test? The site of injection is never really sterile, neither is the needle, when doing a number of tests at one time. Again, it is a well-known fact that the development of tetanus is favored when pyogenic bacteria are introduced into the wound with tetanus spores. I am led to believe so, for last spring I did have a case of tetanus in a cow 21 days following an intradermal T.B. test.

This case report is presented principally for the purpose of ascertaining the opinions of fellow veterinarians on this question.

*Received for publication, September 4, 1937.

SODIUM SULFANILYL SULFANILATE IN CANINE DISTEMPER*

By C. E. LARSEN, *Rushville, Ind.*

Fourteen little dachshunds in a kennel became infected with canine distemper. Serum was used in large doses, repeated every 24 hours for three doses. All made relatively good recoveries but three, which refused all food, became weak, and would not even wag their tails. Having read Dochez' article on treating virus diseases with sodium sulfanilyl sulfanilate, I decided to try it. I gave three 5-grain tablets each day. After the first day, all three pups were up and so much better that we treated them for two more days, each one receiving nine 5-grain tablets. They have all recovered and are playing with the rest. It just worked like magic. I certainly would never be without it again in the treatment of canine distemper and believe it is far superior to anything yet on the market. It is certainly worth trying, as I feel certain at least two of these pups would have died without it.

*Received for publication, April 15, 1938.

ACTINOMYCOSIS IN A HORSE*

By W. F. GUARD, *Columbus, Ohio*
College of Veterinary Medicine
Ohio State University

Subject: Black standard bred race horse, male, four years old.

History: Had distemper when one year old. Shortly afterward, sores appeared in the mandibular region which lasted about two months and then cleared up. There was no further trouble until July, 1932, when sores broke out again on the head and neck. Swellings also appeared, which have been repeatedly lanced. Appetite good, but breathing difficult on account of the swelling on the neck.

Symptoms: Animal dyspneic. Numerous fistulous tracts were present in the submaxillary, pharyngeal and cervical regions. Some sores discharged a clear, serum-like fluid; others, a profuse purulent exudate similar to actinomycotic pus.

The animal was in the hospital eleven days, during which time the temperature gradually arose to 103.2°. Examination of the pus revealed the presence of granules which under the microscope appeared to be typical fungi of actinomycosis. Figure 1 shows the case at the time.

Termination: The animal gradually became more and more depressed and finally died.

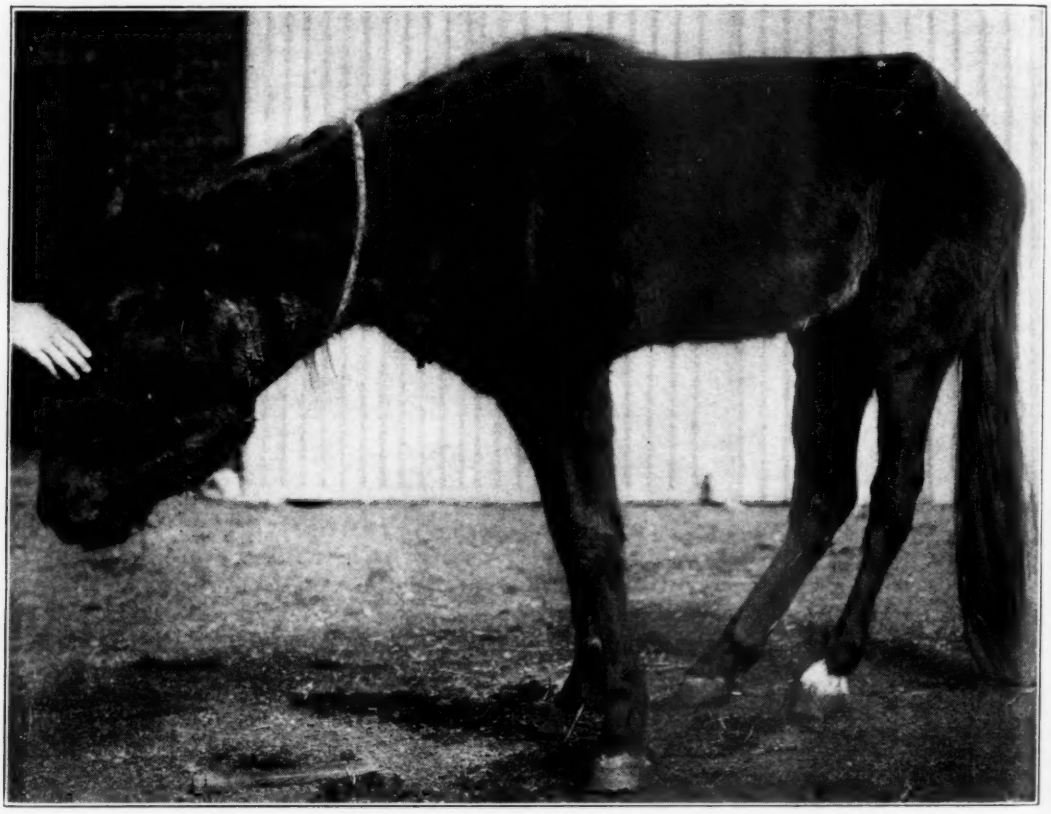

FIG. 1. Actinamycosis in a horse.

Postmortem findings:

1. Pus in several local areas in the submaxillary and anterior neck regions. Abscessed pharyngeal lymph glands.
2. Purulent gangrenous pneumonia.
3. Purulent pleuritis.
4. Fibrinous exudate on surface of liver and diaphragm.
5. Bots in the stomach.

*Received for publication, August 16, 1938.

6. Several round worms in the intestines.
7. Albuminous degeneration of the kidneys.

SEPTIC METRITIS*

By CHAS. HAASJES, *Shelby, Mich.*

A 13-year old purebred Jersey cow had calved normally a number of times and was considered a heavy producer. She had suffered two attacks of milk fever in former years.

On March 18, 1937, the cow gave birth to a calf and did not retain the placenta. About 30 hours later, she went down with typical symptoms of milk fever. The owner attempted treatment by udder inflation. A non-graduate tried a subcutaneous injection. When no improvement was shown, I was called. Examination revealed typical symptoms of milk fever, a vaginal discharge and frequent evacuations of black, fetid, liquid feces. I diagnosed the case as septic metritis. The pus was removed from the uterus which was then irrigated. A quart of mineral oil containing ½ ounce of iodoform and ½ ounce of bismuth subnitrate was injected into the uterus, and stimulants administered. The animal made some improvement, but was so badly emaciated within two months that she was killed. Autopsy revealed an abscess, the size of a coconut, in the liver. The abscess was evidently due to pyemia.

A THREE-FOOT SLIVER*

By CHAS. HAASJES, *Shelby, Mich.*

A team of horses being used for pulling the hayfork were frightened by a cow suffering from nymphomania. The team ran into an old wooden gate. When the animals were untangled, one horse was found to be injured. I was called immediately after the accident. I found that a splinter three-feet long had penetrated the inside of the leg about three inches from the stifle-joint in an upward and backward direction to a depth of more than two feet. It took the combined force of two men to remove the splinter of wood.

After the removal of the piece of wood, the wound was probed with a broom handle which had been disinfected. The probing showed the wound to extend to within about three inches to one side of the base of the tail. An incision was made at this point so drainage and irrigation were made possible. A gauze bandage was drawn through the wound so drainage was assured. A dose of tetanus antitoxin was given and the owner was instructed to irrigate the wound. The wound closed too soon, so an irrigation with a hose with full force of water was used to clean the wound. After that, the healing was rapid and without leaving any noticeable scars.

*Received for publication September 9, 1937.

RUPTURE OF THE UTERUS IN A SOW*

By CHAS. HAASJES, *Shelby, Mich.*

In a shipment of two purebred Duroc Jersey sows from Paducah, Ky., one sow was found dead on arrival at Shelby, Mich. Provisions had been made for the feeding and watering of the animals, but evidently both had been neglected.

At autopsy, the uterus of the sow was found ruptured and seven fetuses found in different parts of the abdomen. The death was attributed to lack of food and water which left the intestines empty, allowing the uterus to become pendulous so when the shipping crate was dropped or jarred, the shock ruptured the uterus allowing the pigs to escape into the abdominal cavity. If the intestines had been full, they, no doubt, would have acted as a cushion to relieve the shock to the uterus.

Unusual

Dr. C. J. Haasjes, practitioner in Shelby, Mich., who has been supplying us with so many case reports, presented an unusual case at the Michigan State Veterinary Medical Association, when an embryonic tooth was removed from a colt. The tooth had become separated from the jaw in its prebirth period and became located at the base of the ear above the eye, according to the report received. Dr. E. F. Meyers performed the operation, after which Dr. Haasjes added it to his growing collection of pathological specimens.

*Received for publication September 9, 1937.

VARIATIONS IN BLOOD TESTS FOR BANG'S DISEASE*

By S. G. COLBY, *Monroe, Mich.*

A four-year-old grade Holstein from a herd of ten cows was kept by the owner for a family cow. This cow (878224) has given birth to two normal calves. The owner stated he had difficulty in getting her with calf, which required several breedings.

On October 22, 1936, the herd was tested for Bang's disease. Nine head sold to a dealer were negative to the agglutination test. Cow 878224 was a reactor, according to the test.

TABLE I—*Tests on cow* 878224

Date of Test	Dilutions				Diagnosis
	1:25	1:50	1:100	1:200	
October 22, 1936	+	+	+	P	Positive
November 2, 1936	+	P	T	−	Suspect
March 12, 1937	−	−	−	−	Negative

+ = complete. P=partial. T=trace. — = no agglutination.

Discussion: Of the nine animals sold, the owner said that one had aborted a year previously. Evidently, the infection was of low virulence, because cow 878224 now has a fine calf about two weeks old. My opinion is that a natural infection in this case produced an immunity, but did not cause abortion.

I am convinced that the inconsistency of the agglutination test is not always due to the antigen or to the personal factor, but to a changing blood picture of the animal. There are several questions which arise from such a case.

1. Is this case an exception or are there many such cows?
2. Can this animal continue to be a carrier of infection and the blood picture be negative?
3. Will cow 878224 continue to be negative?
4. If she is immune, how long will the immunity last?

*Received for publication, March 15, 1937.

TORSION OF THE UTERUS*

By CHAS. HAASJES, *Shelby, Mich.*

The subject of this case report was a four-year-old Jersey cow in her second pregnancy.

Symptoms: When I made my first call, the animal was listless, depressed, off feed, milk supply reduced, and showing colicky pains. Temperature 102.4° F., pulse 83 and respiration 26. Auscultation of the heart revealed no abnormality in that organ. Rectal exploration revealed the fact that the broad ligaments were stretched tightly crosswise over the uterus, which fact interfered with proper palpation of that organ. A slight increase in tension of the uterine arteries was also noted.

Diagnosis: Torsion of the uterus.

A few days later, I was again called to see this cow. She was much worse in every respect and the owner thought that something should be done to alleviate the trouble. I had read that it was a simple matter to perform a laparotomy and reduce a uterine torsion manually.

Treatment: After disinfection and injection of a local anesthetic, the right flank was incised and the torsion located in the vicinity of the pubis. The uterus was found to be so extensively and firmly adherent to the surrounding organs that it could not be torn loose. Therefore, the uterus was incised, the fetus removed and the fluids dipped and siphoned out. Even after this was done, the torsion could not be reduced, at it was not possible to break down the adhesions. Therefore, the animal was destroyed.

In another similar case which I was called upon to treat, postmortem examination clearly showed that reduction of the torsion would have been impossible due to extensive, firm adhesions.

The experiences cited above are given primarily to show that new methods acquired from the literature do not always bring expected results and new ideas should be approached with caution.

*Received for publication, September 9, 1937.

MISCELLANEOUS

Nutrition Conference for Veterinarians

One of the most interesting and enjoyable conferences on the subject of nutrition was held at Decatur, Ind., on August 5, 1938.

That nutrition is a very live subject, especially among large-animal practitioners, is attested by the fact that more than 200 of our most prominent practitioners in Indiana, Ohio and Michigan attended the all-day meeting. Drs. C. F. Huffman, Michigan State College; R. M. Bethke, Ohio Agricultural Experiment Station, and B. H. Edgington, Ohio State University, gave short talks on the feeding of soybean oil meal at the afternoon session.

Both the speakers on the program and the large audience which attended agreed that information volunteered and exchanged at such meetings as these is of great educational value. The soy bean has contributed a large number of derivatives in the past few years which makes it unique as an animal feed. Farm products and the soybean meal, when combined with the proper amount of minerals, makes a very effective and efficient live stock food, according to the testimony of those in attendance. America is just beginning to learn something of the true value of the soybean and its derivatives. As a matter of fact, China has known and used this unusually valuable food for many centuries.

Following the all-day meeting, Drs. J. L. Axby, State Veterinarian of Indiana; F. A. Zimmer, State Veterinarian of Ohio, and C. H. Clark, State Veterinarian of Michigan, gave short talks at a banquet which was held in the evening.

This type of meeting has met with such an enthusiastic response that, no doubt, annual conferences of this sort will be held, not only in Indiana, but in other parts of the United States as well.

Fig. 1. Speaker's Group, Nutrition Conference.

DISEASES OF THE SMALL DOMESTIC ANIMALS. Oscar V. Brumley, V. S., Dean of the College of Veterinary Medicine and Professor of Veterinary Medicine, Ohio State University. 3rd edition. 597 pages. Lea and Febiger, Philadelphia, 1938. Cloth, $4.75.

This is the third edition of a text that has been standard equipment for most veterinary libraries since 1921, when the first edition made its appearance. In order to keep the new edition within proper limits and provide space for new material in the field of small-animal practice, practically all parts of the second edition dealing with poultry diseases have been eliminated. Much of the material in previous editions has been completely revised.

The concise way in which the subject matter is presented will appeal to most veterinary students, but some practitioners would like to see more attention given to the treatment of various diseases. The prophylactic treatment of distemper is covered in two short paragraphs. Considerable space is given to conditions which properly belong under the head of surgery and obstetrics.

Every one of the 597 pages is solid text. There is not a single illustration in the book. The typography is excellent. Both the author and the publisher have done their work well. The book should meet with wide acceptance. H. P. H.

DIAGNOSIS OF PARASITES IN ANIMALS. R. E. Rebrassier, D. V. M., Professor of Veterinary Parasitology, Ohio State University. 65 pages, illustrated. The Ohio State University Press, Columbus, 1938.

Veterinarians generally recognize the importance of correct diagnoses before attempting to treat diseases of animals. This is particularly true of intestinal parasitisms, now that we have a number of drugs that are highly specific as anthelmintics.

Certain laboratory equipment is essential, even for such simple procedures as examining feces and skin scrapings for evidence of parasitism. In this brochure the author lists the equipment needed and then outlines accepted methods of procedure.

There are 121 drawings, photographs and photo-micrographs in the 65 pages. These show the more common parasites, their ova and life cycles. Any practitioner who does his own diagnostic laboratory work should have a copy of this manual. H. P. H.

BOVINE MASTITIS. E. Much-Petersen, M.Sc., B.A., Imperial Bureau of Animal Health, New Haw, Weybridge, Surrey, England, 1938. 272 pages, with 46 tables. Bound, 10s. 0d.

The disease mastitis (mammitis) in cattle has given rise to a wealth of literature, which this book surveys to the end of 1935. Every phase of this subject is covered, and included in its bibliography are the most prominent research workers all over the world, including our own outstanding man of science, Dr. D. H. Udall, who was awarded the Twelfth International Veterinary Congress Prize last year for his contribution to this important subject.

That mastitis is a live and perplexing problem to veterinarians engaged in dairy practice, and one on which the practitioner desires to learn all that he can, is attested by the large crowds that swarmed the mastitis educational exhibit at the A. V. M. A. Diamond Jubilee Convention held in New York, July 5-9, 1938.

To those who would possess a volume containing the most complete review of mastitis that has ever come to the reviewer's attention, and especially valuable for its tables, this book is heartily recommended, with compliments to the author and publisher. E. A. R.

SENSITIZATION AND ANTIBODY FORMATION WITH INCREASED RESISTANCE TO TUBERCULOUS INFECTION INDUCED BY HEAT-KILLED TUBERCLE BACILLI. Jules Freund and Eugene L. Opie. Jour. Exp. Med., lxviii (1938), p. 273.

Rabbits differ widely in the rapidity with which they undergo sensitization with heat-killed tubercle bacilli but, after repeated injections, all animals become sensitized. Intracutaneous injection of a small quantity of heat-killed tubercle bacilli into a previously normal animal produces a nodule which persists from eight to twelve weeks; the same injection into well-sensitized animals produces a lesion which ulcerates within from one to three weeks and is completely healed after about five weeks. Intracutaneous injection of heat-killed tubercle bacilli induces more rapid sensitization than subcutaneous or intravenous injection but, after repeated injections, the difference disappears. Small quantities of B C G induce rapid sensitization and more abundant antibody formation measured by complement fixation than heat-killed tubercle bacilli but, with repeated injections, the difference disappears. Animals that are sensitized and immunized (allergic) before infection are in most instances more resistant to infection than previously normal animals, but there is no correlation between the intensity of sensitization or the titre of antibodies on one hand and resistance to infection on the other. A previously normal animal subjected to infection differs essentially from a sensitized and immunized animal during the first few weeks of infection when sensitization and immunity are developing as the result of infection, but subsequently the progress of sensitization and antibody formation for a time follow the same course. When infection pursues a fatal course, sensitization permanently disappears.

THE ABILITY OF LYMPH TO MAINTAIN VIABILITY IN DEVASCULARIZED LYMPH NODES. Russell L. Holman and Edward B. Self. Amer. Jour. Path., xiv (1938), p. 463.

Popliteal lymph nodes of dogs, when replaced in the popliteal space after complete severance of all vascular and lymphatic connections, rapidly undergo massive necrosis. When all vascular connections were severed with but one or more afferent and one or more efferent lymphatic channels remain intact, infection did not ensue and the nodes remained viable. Chemical analyses of lymph flowing to and from the devascularized nodes showed a sharp drop in reducing substance, bound carbon dioxide and carbon dioxide combining power in the lymph during its passage through the node. These experiments indicate that anaerobic glycolysis is one of the metabolic processes taking place in the viable node. These observations imply a nutritive function on the part of lymph which so far has not been demonstrated *in vivo* in mammals.

EXPERIMENTAL STUDIES ON THE COURSE OF TRICHINA INFECTION IN GUINEA PIGS. The minimum dose of Trichina larvae required to produce infestation of the muscles; with an account of the potential productiveness of the female Trichina. Hans Roth. Amer. Jour. Hyg., xxviii (1938), p. 85.

The experiments indicate that the ration of trichinae encysted in the muscles of guinea pigs per female ingested varies from about 1,000 to 2,500. The potential maximum number of embryos actually delivered by a long-lived female must be considered as being much higher than is indicated by the counts of larvae demonstrated in the digested muscle. Guinea pigs can not only be infested like other laboratory animals by ingestion of minimal doses of

three trichina larvae and upwards, but they proved to be much less resistant to trichinosis as regards the longevity of the adults in the intestines and therefore the coefficient of production of the females, than is the case in other host species (rats, mice, rabbits, dogs and cats). Monkeys alone seem to have somewhat similar ratios to those of guinea pigs.

TRICHOMONAS FOETUS IN BULLS. Justin Andrews and Fred W. Miller. Amer. Jour. Hyg., xviii (1938), p. 40.

The authors describe a method of detecting *Trichomonas foetus* in bulls based upon microscopic and cultural examination of separate ejaculatory fractions from the sheath, seminal vesicles, and ampullae, obtained by intrarectal massage. Immediate microscopic examination of the fractions was more satisfactory than cultural methods. Positive findings occurred most frequently in the seminal fluid and least frequently in the flushings from the sheath. *T. Foetus* was found at autopsy on the *glans penis* and prepuce of three bulls. Organisms were not found in other parts of the genitalia. One bull remained infected for a period of at least eleven months of sexual rest. The authors conclude that the only dependable method of trichomoniasis control in bulls is by preventive methods. This may be accomplished by breeding clean bulls only to unexposed heifers and cows by natural service and by breeding exposed heifers and cows either to infected bulls or to clean bulls by artificial insemination.

INTRAPERITONEAL AND INTRACEREBRAL ROUTES IN SERUM PROTECTION TESTS WITH THE VIRUS OF EQUINE ENCEPHALOMYELITIS. I. A comparison of the two routes in protection tests. Peter K. Olitsky and Carl G. Harford. Jour. Exp. Med., lxviii (1938), p. 173.

Young mice are approximately as susceptible to the virus of equine encephalomyelitis, Eastern and Western strains, when it is administered intraperitoneally, as are adult mice when the virus is injected intracerebrally. With this susceptibility by the intraperitoneal route as a basis, the injection of immune serum-virus mixtures intraperitoneally was found to result in protection in dilutions which give rise to infection after intracerebral inoculation. The difference of protective power by the two indicated routes was shown not to depend on the amount of inoculum nor on the age of the intracerebrally injected mice. Incubation at 37° C. for two and one-half hours neither increased nor diminished the protective action of immune serum when the intraperitoneal method was employed. The phenomenon of selective protection on different tissues was elicited by the sera of hyperimmunized mice, guinea pigs and rabbits, and by sera derived from horses infected with the disease in nature or exposed to it by contact. Of four horses recovered from the malady, all showed antibody in their serum; of others exposed by contact, four of nine animals revealed antiviral bodies when the intraperitoneal technic was employed. The authors conclude that the reaction has significance through its bearing on the mechanism of immunity.

INFLUENCE OF TRANSMITTED LEUKEMIA ON METABOLISM OF UNINFILTRATED LYMPHOID TISSUES. Joseph Victor and James S. Potter. Brit. Jour. Exp. Path., xix (1938), p. 227.

In the intraperitoneal inoculation of a saline suspension of leukemic spleen or lymph nodes of two different mice, transmission lines depressed the anaerobic glycolytic rates but not the respiratory nor aerobic glycolytic rates of uninfiltrated lymphoid tissue. The inoculation of similar amounts of normal spleen or two different strains of mice produced no such effect. No inhibition of anaerobic glycolcysis could be demonstrated when normal and leukemia lymphoid cells were mixed with Ringer solution nor when normal cells were suspended in Ringer solution that had previously bathed leukemic cells. The inhibition of the anaerobic glycolytic activity of uninfiltrated lymphoid tissue of mice which transmitted lymphatic leukemia depends on the following factors: (1) Specific response to individual transmission lines, (2) interval after inoculation and/or quantity of leukemic tissue in the host, and (3) a humoral factor which is either

a product of leukemic cells or the resultant of a reaction between host and leukemic cells.

GLYCOGEN CONTENT OF A FLAGELLATE OF CATTLE, TRICHOMONAS FOETUS. Helen M. Stewart. Amer. Jour. Hyg., xxviii (1938), p. 80.

Trichomonas foetus stained with iodine diffuse glycogen inclusions, the number of which increased with the growth of the individual organism. There was a definite relationship between the consumption of carbohydrates by *T. foetus in vitro* and the accumulation of glycogen within the organisms. The number of organisms containing glycogen increased from the time of inoculation until the maximum number of organisms was reached, and decreased thereafter. The death of *T. foetus in vitro* was always coincidental with the decrease in both numbers and percentage of organisms with high glycogen content. The death of *T. foetus* in serum broth cultures was not due to a deficiency or exhaustion of carbohydrates in the serum, or a combination of the two.

STUDIES ON SODIUM DEFICIENCY. The effects of sodium deprivation on young puppies. Osmo Turpeinen. Amer. Jour. Hyg., xviii (1938), p. 104.

Young puppies, restricted to a ration containing 0.011 per cent of sodium, showed a steady loss in weight, relatively poor appetite and dryness of the skin with a tendency to lose hair. They survived on this diet for eight weeks on the average and died of cachexia. Blood analysis showed a noticeable decrease in blood-sodium values. No marked changes in potassium values were observed. There were no discernible changes in blood calcium, magnesium or inorganic phosphorus. Non-protein nitrogen showed a definite premortal rise, but was otherwise essentially normal.

QUANTITATIVE STUDIES ON GLUCOSE CONSUMPTION BY *Trichomonas Foetus*. Justin Andrews and Theodor von Brand. Amer. Jour. Hyg., xxviii (1938), p. 138.

Trichomonas foetus was proved by direct quantitative methods to consume glucose, as has been inferred by other investigators. The rate of glucose consumption was not uniform throughout the course of a cultural cycle. It averages over 350 milligrams per billion in 24 hours while the average number of trichomonads per 24 hours was increasing. While the average number of trichomonads per 24 hours was decreasing, consumption averaged under 200 milligrams per billion in 24 hours.

QUANTITATIVE STUDIES OF BRUCELLA PRECIPITIN SYSTEMS. II. The precipitation of heterologous antisera by Brucella endoantigens. R. B. Pennell and I. F. Huddleson. Jour. Exp. Med., lxviii (1938), p. 83.

Quantitative cross-precipitation studies with goat antisera show the endoantigens of the Brucella to be serologically distinguishable. Although the endoantigen of *Brucella abortus* and *Brucella suis* are very similar they do not react identically, permitting the serological distinction of the two organisms. These differences in cross-precipitation may be used to identify an organism of the Brucella group or to determine the organism responsible for a Brucella antiserum.

QUANTITATIVE STUDIES OF BRUCELLA PRECIPITIN SYSTEMS. I. Precipitation of homologous antisera by Brucella endoantigens. R. B. Pennell and I. F. Huddleson. Jour. Exp. Med., lxviii (1938), p. 73.

The precipitation by the endoantigens of the three species of Brucella of their homologous antibodies may be described by equations developed from the law of mass action. Since Brucella sera are usually calibrated by determination of agglutinin content and since agglutinins and precipitins have been shown to be identical, the data of the authors imply that the endoantigens may be used for the more accurate calibration of Brucella antisera. The conversely calibrated antisera can be used to detect small quantities of endoantigen. The nitrogen-containing constituent of the endoantigens does not always seem to be intimately connected with the ability to precipitate the specific antibodies.

ARMY VETERINARY SERVICE

Regular Army

Lieut. Col. Wm. R. Wolfe is assigned to duty as attending veterinarian, Lowry Field, Denver, Colo., in addition to his other duties at Fort Logan, Colo.

The promotions of Major Harry L. Watson to the grade of lieutenant colonel with rank from July 18, 1938; 1st Lieuts. Thomas C. Jones and Edwin L. Millenbruck to the grades of captain with ranks from July 24 and July 26, 1938, respectively.

Veterinary Reserve Corps

PROMOTIONS

To 1st Lieut.: J. Vernon Shannon, 244 9th Ave. S., South Saint Paul, Minn.

To Captain: Elmer Fred Finke, 1826 Liberty St., Jacksonville, Fla.

NEW ACCEPTANCES

(First lieutenants)

Anderson, Walter A., Tacoma, Wash.
Anslow, Ralph Owen, Montesano, Wash.
Bankowski, Raymond Adam, Chicago, Ill.
Bach, Clarence Carl, Sebewaing, Mich.
Barber, Russell Albert, New Middletown, Ohio
Baumwell, Earl, Brooklyn, N. Y.
Becher, Ralph Joseph, Botkins, Ohio
Border, Charles Richard, Columbus, Ohio
Bradley, Walter B., Byron Center, Mich.
Brown, Earl Frederick, Manilla, Ind.
Carroll, Howard Francis, Jr., San Francisco, Calif.
Carter, Frank A., Lansing, Mich.
Collins, Wilber Geo., Groton, N. Y.
Cooper, Harold Keim, Poughkeepsie, N. Y.
Covert, Milton Howell, Rochester, N. Y.
Crow, Wilfred Markus, San Bernardino, Calif.
Dawe, Louis Theron, Capac, Mich.
DeGroodt, James Hervey, Mendham, N. J.
Dennis, Walter Roland, Earlville, N. Y.
Eagle, Thomas McCaully, Savannah, Mo.
Elander, Burman John, San Diego, Calif.
Elmore, Robert Geo., Chelan, Wash.
Finch, Dell Clarence, Pullman, Wash.
Firestone, Milton Warren, New York, N. Y.
Fohey, George James, Ann Arbor, Mich.
Francisco, Donald James, Cleveland Heights, Ohio
Friderici, Wm. James, Tiffin, Ohio
Glindmyer, Wm. Edward, Scotia, N. Y.
Green, Geo. Wm., Jr., Scranton, Pa.
Griffith, Robert Lyle, Jr., Inverness, Calif.
Grossman, Henry Earle, Brooklyn, N. Y.
Grossman, Roger, Brooklyn, N. Y.
Halverson, Orville John, Spokane, Wash.
Helyar, James Edwin, Stelton, N. J.
Hirschey, Wilbur Carlton, Castorland, N. Y.
Hulbush, Wm. Russell, Bellingham, Wash.
Hurt, Ross Harrison, Pasadena, Calif.
Jewett, Robert Francis, Cortland, N. Y.
Jimison, Robert Loomis, New York, N. Y.
Johnson, Klemens Frank, Spokane, Wash.
Johnson, Norman Edward, Pullman, Wash.
Kantzer, John Frederick, Bucyrus, Ohio
Kaplan, Sidney Leon, New York, N. Y.
Klar, Jack Milton, Brooklyn, N. Y.
Klooster, Melvin Jacob, Byron Center, Mich.
Krohn, John Ramon, Grandin, N. Dak.
Linn, Frank John, Shelby, Iowa
Lukens, Wm. Lewis, Hillsboro, Ohio
Lusk, Neal Drumheller, Ashtabula, Ohio
McQueen, Geo. Lowell, Vancouver, Wash.
Manly, Philip Charles, Malta, Ohio
Mason, Marcus Maximillian, Pearl River, N. Y.
Maurer, Elmo Laverne, Little Bear, Wyo.
Menaul, Wm., Pasadena, Calif.
Miller, John Wilson, Albany, N. Y.
Moen, Leonard Alfred, Sacred Heart, Minn.
Moody, Robert Albert, Columbus, Ohio
Morehouse, Edward Wray, Salem, Ore.
Mowrer, Robert Francis, Burlington, Wash.
Nagle, Albert Conrad, Woodside, N. Y.
Nichols, Wilbert Charles, Pocatello, Idaho
Ogburn, Leonard Love, Selah, Wash.
Olso, Norman Olaf, Moscow, Idaho
Ott, Hervert Ivan, Wheaton, Ill.
Parker, William Frederick, Berkeley, Calif.
Peters, Stanley Edward, Cleveland, Ohio
Pollard, Morris, Columbiaville, N. Y.
Priddy, Charles Whipple, Los Angeles, Calif.
Raudabaugh, William Gerald, Celina, Ohio
Reichert, Paul Frederick, Chelsea, Mich.
Relken, Walter Edward, Brookside, N. J.
Riker, Joe Thaddeus, San Bernardino, Calif.
Riley, Wm. Francis, Jr., Meriden, Conn.
Robert, Stephen James, Hamburg, N. Y.
Roberts, James Flander, Hamburg, N. Y.
Schiller, Harry, Bronx, N. Y.
Seibert, Norman Edward, Hummelstown, Pa.
Sherwood, Wm. James, New York, N. Y.
Shuman, Richard Duey, Santa Monica, Calif.
Snook, Geo. Williams, Titusville, N. J.
Stevens, Blair Arthur, Pullman, Wash.
Tolley, Robert, Stillwater, Okla.
Tugaw, Edward Anthony, Okanogan, Wash.
Twohig, James Daniel, Seattle, Wash.
Vietti, John Dominick, San Diego, Calif.
Welbourn, Wm. Edward, Union City, Ind.
White, Edward Samuel, Saint Paul, Minn.
Whitehead, Charles Joseph, San Francisco, Calif.
Wright, Alan Wilbur, Smith's Basin, N. Y.
Wright, Donald Albert, Lansing, Mich.
Zook, Roy Firman, Jr., Kansas City, Mo.
Hasson, John Robert, Mulberry, Kan.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

Capt. Howard C. Gale, Arizona Dist., Phoenix, Ariz.

1st Lieut. Ernest L. Henkel, Fort Douglas, Utah

FRED B. ROWAN

Dr. Fred B. Rowan, of Rockford, Ill., died at his home on July 24, 1938, of heart disease, following an illness of 18 months.

Born at Kirkland, Ill., November 23, 1853, Dr. Rowan was graduated from the Chicago Veterinary College in 1890. He practiced for 22 years at Belvidere, Ill., before locating in Rockford.

Dr. Rowan joined the A. V. M. A. in 1921. He is survived by his widow (née Ella Hollingsworth), one sister and one brother.

I. HUSTON BRITT

Dr. I. Huston Britt, of Batavia, Iowa, died on May 4, 1938. Heart failure is reported to have been the cause of death.

Born in Henderson County, Ill., July 13, 1879, Dr. Britt was graduated from the Kansas City Veterinary College in 1908. He had been in practice at Batavia since 1911. He is survived by his widow, one son and one daughter. Six prominent Iowa veterinarians were the pallbearers at the funeral services held on May 6. J. P.

WILLIAM J. HENNESSEY

Dr. William J. Hennessey, of Worcester, Mass., died in Saint Vincent's Hospital, July 30, 1938, at the age of 65 years. He was admitted to the hospital on July 12, 1938, with a stomach ailment.

Born in Fitchburg, Mass., Dr. Hennessey was graduated from the Ontario Veterinary College with the class of 1899. In 1901, he made a trip to South Africa in charge of a transport of horses for the British Army. He started practice in 1904, and had been a resident of Worcester for 33 years.

At the age of 14, Dr. Hennessey was a professional jockey. He was a great lover of horses and had owned and raced many of them. He was president of the Turkey Hill Polo and Riding Club in 1930 and was credited with having introduced polo in Worcester. Dr. Hennessey was a wrestler and boxer, as well as a horseman, and in his youth had competed in the Olympics at Saint Louis in 1904.

Dr. Hennessey joined the A. V. M. A. in 1912. He was a member of the Massachusetts Veterinary Association and served as president for the term 1930-1931. He was a member of the Twelfth International Veterinary Congress and the Worcester Rotary Club. H. W. J.

J. A. FLYNN

Dr. J. A. Flynn, of Jessup, Iowa, died August 6, 1938. In June, of last year, he was operated on and had never fully recovered. His death was attributed to a heart ailment.

Born in Buchanan County, Iowa, January 4, 1888, Dr. Flynn was graduated from the Chicago Veterinary College, with the class of 1917. Following his graduation, he was commissioned as second lieutenant in the Veterinary Corps, July 16, 1918, and was directed to report for duty at the 315th Auxiliary Remount Depot, Camp Jackson, S. C., July 17, 1918. Following services overseas, he was honorably discharged April 9, 1919, and returned to the Chicago Veterinary College, where he took a postgraduate course. At the completion of this period of study, Dr. Flynn established practice at Marcus, Iowa, and a short time later, located at Jessup, where he successfully practiced his profession for over 17 years.

He is survived by his wife, daughter, two sisters and four brothers.

GLEANINGS FROM OUR CORRESPONDENCE

BIRTHS

To Dr. and Mrs. L. M. Becton, of Staunton, Va., a son, Benjamin Valentine, July 27, 1938.

To Dr. and Mrs. Darrel Oldaker, of Fairfield, Iowa, a daughter, Sue Anne, May 10, 1938.

To Dr. and Mrs. E. L. Henkel, of Fillmore, Utah, a son, April 12, 1938.

To Dr. and Mrs. R. B. Mericle, of Omaha, Neb., a son, Robert Bruce, June 4, 1938.

MARRIAGES

Dr. William L. Lukens (O. S. U. '38), of Columbus, Ohio, to Miss Martha Ellen West, of Hillsboro, Ohio, at Columbus, July 12, 1938.

PERSONALS

Dr. Louis C. Dawe (Mich. '38) has started practice in Capac, Mich.

Dr. Willard J. Barga (O. S. U. '38), has opened an office in Dayton, Ohio.

Dr. H. F. Harkavy (K. S. C. '38) has located in Nowata, Okla., for general practice.

Dr. Stephen H. Swain (McK. '09), of Decatur, Ill., recently celebrated his 97th birthday.

Dr. C. F. Runnels (O. S. U. '30) is now located at Clairsville, Ohio, for general practice.

Dr. Forrest McClead (O. S. U. '33) is locating in Huntington, Ind., for general practice.

Dr. John Graf (McK. '14), of Lime Springs, Iowa, is ill at a sanitarium in Saint Paul, Minn.

Dr. E. H. Alspaugh (Cin. '11) is now serving his sixth term as mayor of Wilshire, Ohio.

Dr. J. E. Sargeant (Chi. '20) has purchased the practice of Dr. J. E. Smith, of Fairbury, Ill.

Dr. J. W. Pirie (Iowa '36), of Cedar Rapids, Iowa, accompanied by his wife, are in Kentucky on a vacation.

Dr. Max Peters (O. S. U. '35) has opened an office at Dunkirk, Ind., for the practice of veterinary medicine.

Dr. W. W. Armistead (Tex. '38) is now associated in practice with Dr. W. G. Brock (Ohio '11), of Dallas, Texas.

Dr. D. H. Dickie (Mich. '23) has taken over the practice of Dr. J. A. Schaefer (Gr. Rap. '13), of Bangor, Mich.

Dr. J. J. Strandberg (Chi. '16), of Belle Plaine, Iowa, with Mrs. Strandberg are vacationing in the mountains.

Dr. James D. Nundy (Mich. '38) has located at Bellaire, Mich., following his appointment as Antrim County Veterinarian.

Dr. W. L. Lukens (O. S. U. '38) has entered practice with his father, Dr. Wm. R. Lukens (O. S. U. '12), at Hillsboro, Ohio.

Dr. W. W. Warnock (Ont. '04), of Aledo, Ill., is recovering from serious and painful injuries suffered when kicked by a horse.

Dr. Morris Siegel (Corn. '37), of Spring Valley, N. Y., has become an assistant to Dr. Wesley Laughlin (Ont. '19), of Burton, Ohio.

Dr. E. C. W. Schubel (U. S. C. V. S. '11), of Blissfield, Mich., attended the Nutrition Conference for Veterinarians, at Decatur, Ind.

Dr. M. F. Frevert (Iowa '33), of West Union, Iowa, was confined to his bed in a hospital recently, suffering with a serious infection.

Dr. H. E. Tyner (Chi. '11), of New London, Iowa, accompanied by his wife, are enjoying an extensive motor trip to the Rocky Mountains.

Dr. Wm. J. Pirie (Ch. '18), of Springville, Iowa, had his Minnesota fishing trip ruined in July, by taking seriously sick in the North Woods.

Dr. M. L. Spear (Iowa '31), of Oelwein, Iowa, is recovering from painful injuries suffered when struck in the back muscles by farm machinery in July.

Dr. William Moore (U. S. C. V. S. '11) has opened a modern small-animal hospital of latest design and construction on Route 1, near Raleigh, N. C.

Dr. E. L. Henkel (Wash. '36) reports a change of address from Fort Douglas, Utah, to Fillmore, same state, where he is entering private practice.

Dr. Charles R. Corson (Mich. '38) has taken over a practice of the late Dr. P. E. Schwin, of Elkhart, Ind., and is building a new small-animal hospital there.

Dr. Russell A. Barber (O. S. U. '38) has opened offices in Columbiana, Ohio, where he will conduct a general practice for both small and large animals.

Dr. John F. Kantzer (O. S. U. '38) recently took over the practice of Dr. Marshall F. Douce (Chi. '99), veteran practitioner of Marion, Ohio, upon the latter's retirement.

Dr. R. D. Stewart (O. S. U. '17), of Wren, Ind., and Mrs. Stewart, celebrated their silver wedding anniversary, August 3. He has practiced in Van Wert county since 1917.

Dr. W. W. Warnock (Ont. '04), of Aledo, Ill., was the victim of a serious accident recently, when a colt, which he was treating, turned suddenly, breaking his lower jaw in two places.

Dr. I. C. Shaw (K. S. C. '38), of Staceyville, Iowa, is handling the practice of Dr. B. T. Hartnell (Chi. '12), while the latter is ill and confined to the Mercy Hospital, Rochester, Minn.

Dr. C. M. Prentice (O. S. U. '12), of Clyde, Ohio, has opened a small-animal hospital at 1616 Columbus Ave., Sandusky, Ohio. This will be conducted in addition to his practice at Clyde.

Dr. P. B. McGowan (Cin. '08) was badly burned as a result of an explosion of a can of aviation gas which he had in the rear seat of his car. The gas became ignited by a lighted match.